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An Address.[1]

OCCUPATIONAL DISEASES AND INJURIES OF THE EYE, AND WORKERS' COMPENSATION.

By MARK GARDNER,
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IN discussing industrial diseases and injuries of the eyes, I am going to describe only cases which I have reported on to insurance companies during the past five years. I had kept copies of all reports, 666 in number.

Conjunctivitis.

There were thirty-two uncomplicated cases of conjunctivitis claimed to have been caused by dust, manure *et cetera*, dustmen and men in the building trade being particularly affected. There were ten cases of conjunctivitis associated with keratitis. In one of these it was claimed that the man was directly infected from a pink-eyed sheep. There was a good deal of argument about this case, but the matter was left "in the air". It did not matter much, as it was admitted that the claimant was infected while at his work. Three eyes were left with very little vision as a result of infection from dust from chaff-cutters. Six cases were associated with acute iritis, without any evidence of corneal involvement; in two of these cement was the offending material.

Only in one case was I able to satisfy myself that a persistent conjunctivitis was "self-inflicted". The localization of the inflammatory reaction to the lower bulbar and palpebral conjunctiva suggested the ætiology. In another case conjunctivitis

[1] President's address read before the Section of Ophthalmology, fifth session, Australasian Medical Congress (British Medical Association), Adelaide, August, 1937.

in an eye which had been trephined resulted in the loss of the eye.

Probably a certain number of the corneal lesions mentioned later were preceded by conjunctivitis, which had subsided before any report was made of the case.

Lid injuries were not numerous. Of the eight I reported on, three patients had the lower lachrymal duct cut through, two being due to windscreen injuries and the other to a burst water pipe.

There were eight cases of conjunctival laceration with or without sclera being involved; no disability occurred afterwards in any of these.

Light Ophthalmia.

Cases of light ophthalmia were common. In twenty mild cases short-circuits or oxy-acetylene flare were responsible; more or less severe conjunctival hyperæmia was present, with photophobia.

One case was associated with acute iritis. In another old corneal scars were broken down; in a third case, that of a cinema worker, the ophthalmia persisted and the patient had to give up the job. In only one case was the typical macular lesion seen.

Of considerable interest was the case of a Launceston worker, who, some time after being subjected to a very high voltage flash, began to lose vision. When I saw him about a year later there were extensive *retinitis proliferans*, much vitreous opacity, and diffuse chorioidoretinitis in both eyes. Dr. Bruce Hamilton also reported on him.

Wurdemann reports a parallel case: a motor-man, four months after a flash, showed white streaks in the retina, macular disturbance and later detachment of the retina.

Corneal Conditions.

I have noted 324 cases as primarily corneal conditions; in 42 abrasion was present with no considerable opacity resulting; in 12 there was keratitis, mostly of disciform type, following abrasion. In most of these there was considerable loss of vision.

There were 230 cases in which foreign bodies were involved; in the majority no substantial loss of vision occurred; 26 were associated with hypopyon ulcer, mostly due to stone or coal; in only two of these was lachrymal mucocele present. In 14 there was extensive keratitis, with much loss of vision. (These were not the hypopyon cases, which, of course, all had bad endings.)

Burns.

I have reports on 69 cases of burns. Molten metal was easily the commonest cause, there being 23 cases. Lime was responsible for eight, caustic soda for seven, ammonia for five, spirits of salts for five, hot tar for four, sulphuric acid, sulphur dioxide, coal gas and hydrochloric acid for two each, and nitric acid, nitro-glycerine, kerosene, ferric chloride, boiling soap, hot fat, crude oil, steam and lithophane for one each.

The worst results were those caused by ammonia, sulphur dioxide and nitric acid.

Perforating Injuries.

Perforating injuries numbered 104. There were eight perforating wounds of the cornea, with no prolapse of the iris or cataract, six with prolapse of the iris and no cataract, eight with prolapse and cataract, 15 with no prolapse but cataract, one with an eyelash carried into the anterior chamber, six perforating wounds of the sclera. There were 60 intraocular foreign bodies. In 18 no cataract was noted; 50% of these eyes were lost or had no useful vision. Three cases of siderosis were reported on.

Detachment of Retina.

There were 15 cases of detachment of the retina. In two, in which injury at work was claimed, neoplasms were found. In five the affected eye was myopic. Ten were operated on; three completely successful results were noted.

Ruptured Globe.

In twelve instances the globe was ruptured. Two ruptures were caused by our national weapon, the beer bottle, and two by a cow's horn.

One patient, a young man, with only one eye, which was extremely buphthalmic, worked in the Blind Institute; his vision was about $^2/_{\infty}$. While playing cricket with the blind team, his eye was struck and ruptured by the ball (made of wicker, with a bell inside).

Dislocation of the Lens.

Dislocation of the lens occurred in nine cases.

Only two of these were caused in the usual way, during wood-chopping. My experience in hospital is that the majority of cases of dislocation of the lens are due to pieces of wood flying up; but as the accident generally happens at home there is no claim for compensation.

Rupture of the Chorioid.

Rupture of the chorioid occurred in four cases.

In one of these which I saw recently, the injury was caused by a bursting emery wheel; the other eye was aphakic, as a result of a perforating injury some years previously, and had very little vision. The rupture, unfortunately, was between the optic disk and macula.

Vitreous Hæmorrhage.

In nine cases vitreous hæmorrhage was present. In four the result was bad, no useful vision being recovered. The other cases cleared up. I reported on only two cases of contusion cataract; no case of occupational cataract occurred.

Hole at the Macula.

In two cases a hole at the macula could be ascribed to recent injury.

Muscle Paralysis.

Muscle paralysis occurred in 13 cases; six were associated with fracture of the skull. Types of direct injury to muscles were inferior rectus paralysis after a blow from a cow's horn. The superior oblique muscle was paralysed through a blow from a horse's head. The superior rectus was paralysed in two cases; the injury was caused by a hook in one, and in the other by a splinter of wood. This last

patient was under my care at the Eye and Ear Hospital; tenectomy of the opposite inferior oblique muscle relieved the diplopia.

Other Conditions.

In one case the poor vision of an emblyopic eye was claimed to be the result of trauma. I have reported on four cases of simulation of blindness after minor injuries. Twelve explosion injuries were due to gelignite, mine blasting, gas and gun powder; the damage in most of these was considerable. Four cases of optic atrophy followed fracture of the skull.

Discussion.

From my experience I would suggest that in about 90% of the cases sent by insurance companies for opinion and report, there is no doubt that the ocular condition present can be ascribed to the alleged injury or infection incurred during the course of the employee's work.

Cases requiring careful consideration are those in which some trauma is claimed to have occurred and typical interstitial keratitis is present, or there is a flare-up of old trachoma, or chronic irido-cyclitis develops, with a "+" response to the Wassermann test. It must be remembered, however, that when there has been definite evidence of some injury at work and such injury has precipitated an interstitial keratitis, the condition must be ascribed to the injury. I recall two cases in London in which I gave evidence in court, in which this was established.

Some cases of keratitis present difficulties; perhaps a week or so after some trivial injury, which may or may not have been reported at the time, a keratitis develops, and the employee may be out of action for a very long time; our insurance friends are sometimes a little surprised at our reports on such cases, if we find for the plaintiff.

A worker develops an acute catarrhal conjunctivitis; he is bound to remember getting some dust in his eye; we cannot always be sure about such cases.

I have already mentioned a case of amblyopia and cases of simulation of blindness. In other cases in which a claim has been made that conditions arising out of employment have been responsible for loss of vision, I have found chronic glaucoma, pituitary tumour and specific paralyses.

A worker, some months after an admitted injury, was found to have detachment of the retina in the eye concerned. Operative treatment was not successful. At the time it was noted that the other eye was moderately myopic and had vitreous opacities. He went back to his job, and turned up at the hospital later with a detachment in his good eye. In his job he had to do some lifting at times, and it was claimed that the conditions of his work were responsible for the detachment.

Well, he may have got it carrying out the rubbish tin at home, or picking up the baby, or from being jolted in a tramcar. One of the patients on whom I reported got his detachment by being hit on the eye by a falling peach. There is plenty of room for argument in such a case.

Workers' Compensation Acts.

I shall now briefly discuss the *Workers' Compensation Acts* of the different States in their application to eye conditions. In all the Acts, compensation for disabilities is in the form of a lump sum; in this respect our Acts differ from many of the European Acts, which provide compensation in the form of a pension, based on the estimated loss of earning capacity occasioned by the injury. In Victoria, New South Wales and Western Australia definite sums are laid down for the different disabilities; in South Australia, Queensland and Tasmania sums are stated as percentage ratios of compensation to full compensation as for total incapacity; this amount varies according to the past earning capacity of the worker concerned.

In the Acts of the three States first mentioned, and also in the Commonwealth Act, there is a very important addition, which allows compensation for partial loss of sight of both or one eye.

In the amended Victorian *Workers' Compensation Act* the following are the provisions for compensation of eye disabilities:

Total loss of sight of both eyes ..	£750.
Total loss of sight of only eye ..	£750.
Total loss of sight of one eye, together with the serious diminution of sight of other eye	£562 10s.
Total loss of sight of one eye ..	£300.
Partial loss of sight of both eyes	Such percentage of £750 as is equal to the percentage of diminution of sight.
Partial loss of the sight of one eye	Such percentage of £300 as equals percentage of diminution of sight.

When consequent upon injury to a worker a cataract is removed from one or both eyes, there shall be taken into account in assessing the compensation payable the actual diminution of sight and the loss of binocular vision occasioned by the removal of such cataract, and when a worker suffers injury to an eye which results in the formation of a cataract and it is deemed inadvisable to remove such cataract, the compensation payable to such a worker shall be the compensation payable for the total loss of the sight of one eye, and if the worker subsequently suffers an injury whereby he loses the sight of the other eye, the compensation payable therefor shall be such percentage of £300 as would be equal to the percentage of the diminution of sight of the first injured eye after the cataract was removed.

None of the other Acts in the States have these two last clauses.

In some of the Acts this note is added: total loss of one eye shall be deemed to include the permanent total loss of the use of such eye; but even if this is not expressly laid down, we may take it for granted that if we report to the effect that an eye has no useful vision for industrial purposes, all

companies will accept that as equivalent to the total loss of the sight of the eye.

How are we to estimate percentage diminution of vision?

The estimation of the degree of incapacity resulting from eye injuries is a delicate and complicated task involving a judgement relating the condition of the eyesight of the individual concerned with all the other circumstances which affect his capacity in his old occupation and his ability to enter a new one. We have the simpler task of merely deciding what ratio a certain loss of visual acuity bears to a total loss of vision, and have not to worry about earning capacities.

Many American States have tables for measuring visual efficiency for compensation purposes; that of Dr. Black, prepared for the American Medical Association, is the only one concerned with any function other than visual acuity. In this, binocular vision and field of vision are taken into account, 25% being allowed for loss of binocular vision, ratios of 50% for loss of central visual acuity, and 2·5% for each reduction of 5° of field of vision between 60 and 5.

No European workers' compensation law describes a scale of injuries and corresponding percentages of incapacity, but tables based on medical opinion are commonly used as guides by those responsible for evaluating incapacity.

Complicated formulæ have been devised in some countries to estimate loss of earning capacity related to loss of vision *et cetera*. I do not propose to discuss them. Fergus has stated that it is impossible to reduce to mathematical formulæ anything involving human action based on individual volition and intelligence. All people whose vision is reduced, say, to $^{6}/_{36}$, will not be of equal capacity as regards work; in some forms of work visual acuteness is of primary importance—watch-making, for example; in others binocular vision may be essential. In manual labourers, efficiency depends largely on light sense and sense of alignment and binocular vision in most cases is not essential. Nearly all kinds of work can be done efficiently with one eye, but it is not too easy for a one-eyed man to get a fresh job.

After the Victorian *Workers' Compensation Act* had been amended to allow compensation for partial loss of sight, at a meeting of the Ophthalmological Section of the Victorian Branch of the British Medical Association I suggested the following scale of percentages:

In unskilled workers, reduction of vision to	$^{6}/_{12}$ equivalent to 10%
In unskilled workers, reduction of vision to	$^{6}/_{18}$ equivalent to 25%
In unskilled workers, reduction of vision to	$^{6}/_{24}$ equivalent to 50%
In unskilled workers, reduction of vision to	$^{6}/_{36}$ equivalent to 60%
In unskilled workers, reduction of vision to	$^{6}/_{60}$ equivalent to 80%

That this estimation of "age reduction of vision" should not be regarded as absolute, but should serve as a guide in assessment of visual incapacity for compensation purposes. In the case of skilled or clerical workers, near vision to be also taken into account.

That 50% be allowed for loss of binocular vision apart from percentage allowed for loss of visual acuity (with correcting glass).

That where there is loss of sight in both eyes the estimation be made on a basis of two-thirds of the added percentages.

That when there are losses of field of vision, or permanent muscle paralyses, no percentage scale be drawn up, but every case to be considered on its merits.

That three months should elapse after the last evidence of inflammatory reaction before amount of visual incapacity is estimated.

These proposals were accepted by the members; I should like to hear what you think, and invite criticism.

We did not come to any decisions as to whether we are to measure the visual acuity with correcting glasses. Wurdeman quotes a judgement in the United States of America:

There is nothing in the Act indicative of any intention that a corrective lens should be considered in determining the loss of the fractional part of the vision of the eye.

I should think the same would apply to our Acts.

The nature of the employment or the type of correcting glass necessary may make it impossible for glasses to be worn; then I think that we should estimate the visual acuity unaided.

Recently I examined a man who had had a perforating wound at the limbus; a large piece of metal was removed. When everything settled down, his vision was $^{6}/_{6}$ partly, but a −5 dioptre cylinder was necessary.

You will agree that it would be wrong to say that the visual capacity in the damaged eye was 100% efficient.

I am quite prepared to find that some of those present think that the vision of a labourer reduced to $^{6}/_{18}$ is over-assessed at 25%; in the Victorian Act that would represent £75 compensation; but he would get £90 if he lost one of his little fingers.

I am strongly of the opinion that 50% is not too much assessment for loss of binocular vision in cases of traumatic cataract. Our experience with perforating wounds gives us cause to think that an eye which has had a foreign body roaming about in the vitreous may not always stay good. No doubt, most unskilled workers can get on quite well with their job with one good eye and the other aphakic, but there are other things besides work: a man's hobbies and recreations may be seriously interfered with, and I personally would feel that having to read with one eye for the rest of my life, after being accustomed to the use of two, would entitle me to as much compensation as a man who had lost one of his big toes; or to twice as much compensation as a man who had become deaf in one ear; or to even four times as much as a worker who had lost any one of his other toes, or a joint of a finger. Would any of you employ a chauffeur with an aphakic eye?

So if we allow 50% compensation (of one eye) to a man whose vision has been reduced to $^{6}/_{24}$, I

think it is logical to allow 50% for the aphakia, and if vision is only $^6/_{24}$ with the best correcting lens, to allow another 25% for that reduction.

My aim in writing this paper was to confine myself to practical issues, and I invite criticism, kind or unkind; I feel sure that many points of interest will be brought out in the discussion which follows.

PUERPERAL INFECTION DUE TO HÆMOLYTIC STREPTOCOCCI.[1]

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PUERPERAL sepsis is due to the invasion by pathogenic organisms of the raw wounds that remain after parturition or abortion. At whatever stage of gestation the uterus delivers its contents, the placental site remains as a most favourable area for the multiplication and dissemination of an invading organism. During most deliveries the cervix has been torn and the vaginal mucosa has suffered lacerations of varying degree. The entry of organisms through these areas is responsible for a large and constant proportion of maternal deaths and is a major cause of morbidity after child-bearing. Table I shows the number and percentage of maternal deaths due to sepsis in three large investigations, and Graph I indicates that the death rate in Australia has remained unaltered for many years.

When a search is made for organisms in the genital tract of infected women admitted to the hospital, it has been found that in the more severe cases usually either the hæmolytic streptococcus, the anaerobic streptococcus or the *Bacillus coli communis* is present in large numbers; less frequently other organisms, such as *Staphylococcus aureus*, non-hæmolytic streptococci, pneumococci *et cetera* have been isolated. Table II shows that the hæmolytic streptococcus is found in nearly 40% of these more severe puerperal infections and that it is responsible for about 80% of the deaths from sepsis.

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

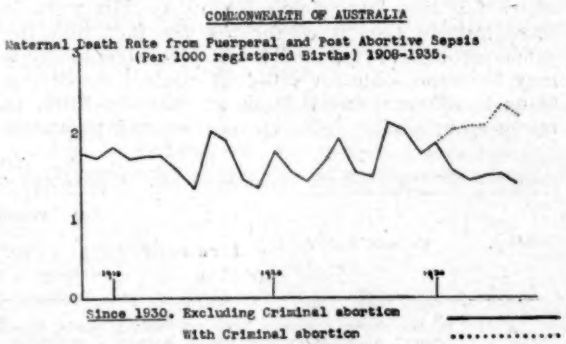
TABLE II.

Bacteriological Examination of Puerperal Infections.

Observation.	Number of Cases Examined.	Hæmolytic Streptococcal Infections.	Percentage Mortality of Hæmolytic Streptococcal Infections.	Percentage of Cases Infected with Hæmolytic Streptococci in all Deaths from Sepsis.
E. B. White (1927)[4] ..	53	24 (45%)	—	—
J. Smith (1931)[5]	196	149 (76%)	30·0	93·0
Colebrook *et alii* (1933)[6]	533	208 (39%)	26·0	73·0
Queen Charlotte's Hospital Report (1935)[7]. ..	270	95 (35%)	25·0	86·0
Rankin (1933)[7] ..	82	36 (46%)	37·5	81·8

It is obviously the most important organism both in its incidence and virulence.

Early investigators of vaginal bacterial flora, such as Burt White and Armstrong,[9] and Taylor and Wright,[10] found hæmolytic streptococci in



GRAPH I.

the cervix of a small proportion of women before labour, yet these very women were free of serious infection after delivery, whilst other women who were not harbouring these organisms ante-natally developed severe puerperal infection and hæmolytic streptococci were found to be present. Nor were

TABLE I.

Observation.	Number of Deaths Investigated.	Deaths from Sepsis Following Full Time (or Premature) Labour.	Deaths from Sepsis Following Abortion.	Total Deaths due to Sepsis Following Abortion and Labour.
N.S.W., 1929–1933[11]	1,073	167 (15·6%)	169 (15·6%)	336 (31·0%)
Departmental Committee (British), 1933[12]	4,655	1,729 (37·1%)	Not stated.	Not stated.
New York City (U.S.A.), 1930–1933[13]	2,041	510 (25·0%)	262 (12·8%)	722 (37·8%)

these investigators able to differentiate between the strains recovered. The explanation of this became apparent with our increasing knowledge of the complex family of streptococci. In 1933[11] Rebecca Lancefield devised a precipitin test by means of which she was able to separate into distinct and sharply defined groups the streptococci giving an area of hæmolysis on blood agar plates and to show that all those found in pathological conditions in man belonged to one group which she called A. Table III shows our present knowledge of these groups.

In 1934 Lancefield and Hare[12] examined and placed in their serological group the strains of hæmolytic streptococci recovered from the cervix or upper part of the vagina of women in Queen Charlotte's Hospital, London. With one exception the strains from severe puerperal infections (46) were identified as members of Group A. The few strains recovered before labour from the birth canals of 855 of the women were not of Group A. Eighteen strains not belonging to Group A (mostly B and D) were found in patients with minor degrees of infection.

Meanwhile Griffith[14] had been studying serologically strains recovered from the various types of streptococcal infections in man. He has found twenty-seven serological types, nearly all of which fall into Lancefield's Group A. His work is important in that it establishes the fact that the same serological type of hæmolytic streptococcus may be responsible for different clinical manifestations in different individuals, so that tonsillitis in one person, scarlet fever in another, and puerperal sepsis in a third may be all due to an identical organism.

It may now be stated with reasonable certainty, firstly, that pathogenic hæmolytic streptococci do not normally inhabit the female genital tract, secondly, that when pathogenic hæmolytic streptococci do gain access to the genital tract of parturient women they nearly always produce a pathological state.

For practical purposes it may be assumed that when an hæmolytic streptococcus is found in association with puerperal fever to 101° F. it belongs to Group A and is consequently dangerous to others.[1] In addition it indicates an extrinsic source of infection, and this should be looked for when possible.

Investigations at the Women's Hospital, Sydney.

I now propose to discuss the results of 286 cervical swabbings taken from patients in the Women's Hospital, Sydney.

There are two groups: (i) Those taken on the third day of the puerperium from 100 consecutive women delivered in hospital. (ii) Those taken from the cervix of women delivered in hospital or admitted to hospital after delivery outside, or suffering from abortion, who sustained a temperature of 101° F. for which no extragenital cause could be found.

[1] During the discussion which followed the reading of this paper it was pointed out that a few cases of puerperal infection due to hæmolytic streptococci have been reported in which the organism did not belong to Group A. In our series the organism found was grouped in thirty-one cases. All but three belonged to Group A, and these three were associated with minor degrees of infection.

TABLE III.

Known Serologic Groups and Types of Hæmolytic Streptococci.[13]

Group.	Probable Normal Habitat.	Probable Secondary Habitats.		Type Differentiation.
		Associated with Infections.	Not Associated with Infections.	
A	Human carriers. Human infections: Scarlet fever, puerperal sepsis, erysipelas, septic sore throat, pneumonia *et cetera*.	Mastitis in cattle, the infection probably originating from human beings.		Divided into serological types on the basis of specificity of *M* substance (Lancefield) or slide agglutination (Griffith). 27 types.
B	Cattle mastitis, milk.		Human throat and vagina.	Divided into four or more types.
C	Horses, strangles, endometritis. Cattle, mastitis. Guinea-pigs, adenitis. Also infections of many other animals as rabbits, foxes, swine, fowls.		Human throat and vagina.	Tentatively divided into four types.
D	Cheese.		Human throat, intestine and vagina.	
E	Milk.			Serological types known to exist.
F	Man, respiratory tract.			
G	Man, respiratory tract. Monkey, normal throat pneumonia. Dog, otitis.			

Other groups known to exist.

TABLE IV.
Cervical Swabbings Taken on the Third Day of Puerperium.
(100 consecutive deliveries.)

Number with Febrile Puerperium. (Temperature, 101° F.)	Hæmolytic Streptococci Found.	
	Febrile.	Afebrile.
11	4	1

The strains from the four patients who developed a febrile puerperium reacted to the precipitin test for Group A, while the one strain from the afebrile patient did not. Although small, this series acted as a control and the results were as anticipated.

TABLE V.
Table showing the Incidence of Morbidity (Temperature 101° F.) and of Hæmolytic Streptococcal Infection in Three Groups of Hospital Patients.

Admissions During Eight Months.	Number with Temperature 101° F. During Puerperium.	Number with Hæmolytic Streptococci in Cervical Swabbing.	Incidence.
Consecutive deliveries in hospital: 1,900 ..	122 (6·5%)	31 (25% of swabs taken).	1·6%
Patients admitted after delivery of viable child: 49	20	9 (47% of swabs taken)	19·0%
Patients admitted with abortion: 363	51	4 (8% of swabs taken).	1·1%

Two things call for comment:

1. The incidence among consecutive deliveries is six times greater than the 1 in 400 that Colebrook[15] regards as a standard to be attained, and one that has been attained by at least three British hospitals. (Quoted by Dora Colebrook.[16])

2. The incidence among abortions is low. It would appear that although infection is much greater after abortion than labour (as is to be expected), it is much less likely to be due to the hæmolytic streptococcus. This is supported by consideration of the organisms causing death in each case group (see Table VI). To this series may be added a further thirteen consecutive cases of hæmolytic streptococcal infections, which were collected before routine swabbings were instituted.

Ætiological Factors.

Nature of Labour.—More than half of the patients had had a normal delivery, and a large proportion of the remainder only forceps delivery or a perineal rupture.

Parity.—Analysis of this series of cases shows that parity has no bearing on ætiology. The number of infected patients in each parity group is proportional to the number of women in each group.

Age.—The average age of the infected patients is five years greater than the average age of all the women delivered during the period reviewed.

Day of Onset of Fever in Puerperim.—The characteristic onset is a sudden rise of temperature, usually 102° to 105° F. on the second, third or fourth day of the puerperium. This fact suggests that the infection takes place at the time of delivery rather than later.

Reference to Graph II shows that onset of fever over temperature of 101° F. on these days is most likely to be due to streptococcal infection. As lactation is being established during this time, some

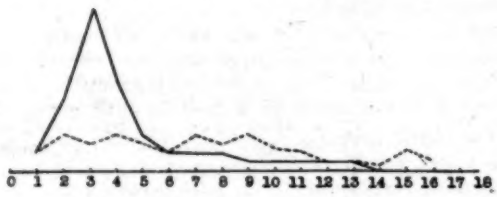
TABLE VI.
Total Series of Hæmolytic Streptococcal Infections.

	Number.	Severity of Infection.[1]	Deaths.	Deaths from Infection by other Organisms during Period Reviewed.
Among women delivered in hospital ..	36	Grade I .. .. 31 Grade II .. .. 1 Grade III 4	0	0
Among women admitted after delivery of a viable child.	17	Grade I .. .. 11 Grade II .. .. 2 Grade III 3	4	1
Among women admitted with abortion	5	Grade I .. .. 3 Grade II .. .. 2 Grade III 0	0	*Bacillus coli communis* .. 3 *Staphylococcus aureus* .. 1 Hæmolytic *Staphylococcus albus* 1
Total	58		4	

[1] Summarized:

		Cases.	Deaths.
Grade I.	Infection limited to uterus, vagina and perineum	45	0
Grade II.	Infection extrauterine but localised to pelvic cellular tissue ovaries, tubes or pelvic peritoneum	5	0
Grade III.	Septicæmia with or without generalising peritonitis or/and involving heart or lungs..	7	4

cases of so-called "milk fever" may in reality be due to this infection of the genital tract. Sometimes pain and tenderness in the lower part of the abdomen and iliac fossæ is present. If the infection remains localized to the uterus—depending on the resistance of the patient, the virulence of the organism, and the size of the infecting dose—the temperature usually remains high for from two to five or six days, subsiding by lysis or sometimes abruptly. This type of infection is, I think, usually unrecognized as a streptococcal one, yet the patient requires isolation and a possible source of infection should be looked for. If the organism invades beyond the uterus, the patient is more gravely ill, and diagnosis is made by blood culture and recognition of clinical signs on general and pelvic examination.



GRAPH II.

Graph showing comparative frequency of day of onset of fever reaching 101° F. and over. The continuous line represents hæmolytic streptococcal infections; the dotted line represents other puerperal genital tract infections.

Treatment.

Good nursing, plenty of fresh air and adequate attention to such matters as regular bowel action, sleep, diet and relief of headache or pain are of utmost importance. Large doses of a concentrated vitamin *A* preparation, such as "Radiostoleum" (one drachm twice a day), may assist in increasing natural resistance. A full blood count should be made, as secondary anæmia is commonly associated. A high leucocyte count, if maintained, is of good prognostic omen. A low red cell count and hæmoglobin value are indications for blood transfusion, and since owing to the hæmolysis the improvement in the red cell count is often only temporary, blood transfusions of from 400 to 600 cubic centimetres of blood should be repeated if necessary every few days. Useful adjuncts are iron and liver therapy. Of the administration of sulphonamide I shall speak later.

While the infection is localized to the uterus, leave well alone, unless the lochia is offensive or uterine drainage inadequate, perhaps from closure of the os or retroversion of the uterus. In these circumstances the uterine cavity may be irrigated with two ounces of glycerine every six hours, introduced through a self-retaining catheter, which is left *in situ* for twenty-four hours.

The insidious onset of generalizing peritonitis must be watched for. There is no diagnostic clinical syndrome, and it is the most fatal complication.

Thirty fatal cases of peritonitis at Queen Charlotte's Hospital[6] exhibited the following symptoms:

Tympanites	22
Tenderness	13
Pain (with or without tenderness)	9
Rigidity	4
Diarrhœa	5
Vomiting	5
Free fluid (clinical examination)	2

In two cases there was absolutely no suspicion of general peritonitis until *post mortem*.

Associated with a dry coated tongue, raised temperature and rising pulse rate, the presence of any two (or at most three) of these signs or symptoms is indication for exploration and drainage. This may be done through the abdominal wall or the posterior fornix, and if free fluid containing pus cells is found, provision should be made for adequate drainage.

Continuous intravenous drip of 5% glucose in saline solution should be instituted at once in the hope of promoting a flow of fluid into the peritoneal cavity. Since the introduction of sulphonamide the treatment of hæmolytic streptococcal peritonitis at Queen Charlotte's Hospital is mainly medical.

Intravenous chemotherapy with antiseptics such as mercurochrome has had its day, but the custom of administering specific antisera dies much harder. Rankin[17] and Colebrook[18] have each published their results in large series of patients with untreated controls, and using both puerperal antistreptococcal sera and scarlet fever antitoxin. In each series the mortality was least among the controls. My own observation of serum-treated patients has satisfied me that it is of little value, although if the patient has a scarlatinoform rash, scarlet fever antitoxin may be tried.

The discovery of a new chemotherapeutic substance sulphonamide and allied forms has given new hope in these conditions. You are all no doubt familiar with the experimental work that has been done on animals and humans. Forty-one of our fifty-seven patients were treated with sulphonamide preparations. Of the forty-one patients, four died, a mortality rate of 10%. This series is small, and, as we are unable to compare it with a similar series of untreated patients, the results must be regarded as inconclusive.

The more severe cases came early in the series when the dosage may have been inadequate in view of later knowledge, and, owing to the impossibility of getting regular supplies of any one preparation, various preparations have been used. Early in the series we used "Prontosil Red" and "Prontosil Soluble", then "Prontosil Album", and latterly "Proseptasine" and "Streptocide".

Of the four patients who died, three had extensive infection on admission to hospital, one with well-marked general peritonitis and septicæmia, one with malignant endocarditis with commencing gangrene of foot, and the third with septicæmia and bronchopneumonia. It could hardly be expected that any

therapeutic agent could overtake such advanced conditions.

There are some human pathogenic strains which are not virulent to mice, and the fourth patient who died was infected with such a variant. As all the experimental work on mice protection has of necessity been done with high mouse-virulent strains, it is not known whether sulphonamide has any effect experimentally on non-mouse-virulent strains such as this was. In twenty-eight cases the infection clinically was localized to the uterus. Many of these patients were very ill; in others the infection was mild. It is not possible to say whether sulphonamide was responsible for preventing extension from the uterus, but in many instances improvement seemed to be concurrent with its administration. In several cases a relapse and recurrence of high temperature has followed some forty-eight hours after withdrawal of the drug and has quickly subsided again after its readministration. Of the remaining eleven patients, in whom the infection spread beyond the uterus, three had positive blood cultures and all recovered. My impression from watching these cases is that if the infection is recognized early as due to a hæmolytic streptococcus and the patient receives adequate doses of sulphonamide, in addition to good general treatment, the frequency of the invasion spreading beyond the uterus with its resultant high mortality would be greatly lessened. Recently a test has been devised by Marshall and others[19] for determining the presence and concentration of sulphonamide in the blood and urine. It has been found experimentally that the drug is rapidly absorbed from the gastro-intestinal tract and that concentrations between 1 in 5,000 and 1 in 10,000 can be attained and maintained in the blood. Excretion by the kidney is rapid (except in renal inefficiency) and nearly 100% of the drug administered may be recovered after equilibrium between intake and output is established. To reach a high blood concentration give a single large dose (0·05 gramme per kilogramme), and in eight hours commence giving the daily dose of 4 to 6 grammes equally divided into four-hourly intervals.

Excretion is delayed if renal impairment is present and the dose should be decreased.

Indications of Toxicity.—Nearly every patient became blue, some very blue, owing to the presence of methæmoglobin or sulphæmoglobin in the blood. The workers of Queen Charlotte's Hospital regard it as of no consequence, but if the cyanosis is very intense the dose is reduced. The blueness disappears within a few days after ceasing administration and the patients are apparently unaffected. We had one exception to this in a patient with a sensitivity to this drug who developed extreme cyanosis and raised respiratory rate after a small dose of "Prontosil Album". It was some weeks before the blueness disappeared. A few patients have complained of severe headache, and one vomited the drug persistently. This has also been observed at Queen Charlotte's Hospital, and the difficulty has been met with intravenous drip administration (eight grammes a day).

Prophylaxis.

Immeasurably better than cure is to prevent the infection. This rests with the patient herself and with her attendants during delivery and puerperium. The patient must be told of the risk of contact with other infections due to the same organism, scarlet fever, erysipelas, *otitis media*, quinsy *et cetera*. The doctor and nurse at the confinement must satisfy themselves that they are not a source of infection. Except in large teaching institutions, masking is an exception rather than a rule, whereas it should be a *sine qua non*, as in other major surgical procedures. Should the patient have an abrupt rise of temperature to 101° F. early in the puerperium, a swabbing from the upper part of the vagina is necessary. The procedure is simple and should be as much a routine as a throat swabbing in suspected diphtheria. The organization of laboratory facilities to culture these swabbings is a matter for health departments. It should not be difficult to have listed the available laboratories capable of doing this and allotted to serve defined areas.

Immediate notification of a positive finding should be made to the health authorities and they should then see that the patient is adequately isolated and treated, and should look for a possible source of infection. The other organisms causing sepsis are practically all autogenous and are not dangerous to others. They do not point to a dangerous outside source of infection and their care can be left to the judgement of their medical attendant.

The law in New South Wales at present requires notification of all puerperal pyrexia. In my opinion, much better results would be obtained if notification of streptococcal infections only was asked for. This would stress the significant and important difference between streptococcal and non-streptococcal infections and compliance with the law could be more easily enforced than at present. The medical practitioner would soon realize the diagnostic value of swabbing the cervix of any puerperal patient with a temperature of 101° F. The comfort of finding it "negative" is well worth the trouble. With the present system, doctors, nurses and hospitals tend to take a chance on puerperal pyrexia subsiding, with the result that adequate isolation and treatment are often left too late. It is well recognized that early removal to hospital gives a better chance of recovery to the patient. With willing cooperation between the medical and nursing professions and the health authorities, and intelligent application of our present knowledge of the ætiology of puerperal infections, I believe that the maternal mortality rate of Australia could be reduced by 1 per 1,000.

May I conclude by quoting from the report of the Medical Research Council[20] for the year 1935-1936.

> It is quite certain that we ought not to rely in the first place on curative drugs. Clearly the ideal to be aimed at

in any disease is to prevent its development and thereby make alleviating remedy unnecessary. This is especially true as regards disease associated with the natural process of childbirth. Modern investigation suggests two lines of prevention. First, childbirth should be so conducted that access of pathogenic organisms to the birth canal of women must be vigorously excluded, especially streptococci associated with sore throat, whether in the respiratory tracts of medical attendants, or of friends, or of the patients themselves. Secondly, the diet of pregnant women should be of such a nature as to raise the resistance to infection to the highest possible level: this means an ample consumption of milk, green vegetable and other protective foods. If for any reason, known or unknown, these first two lines of defence are broken down and the puerperal sepsis does develop, then is the time to bring into action the third line of defence, namely, the treatment which evidence suggests is now available.

Acknowledgements.

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THE SOURCE OF INFECTION IN PUERPERAL SEPSIS.[1]

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GRIFFITH[1] has said that the control of scarlet fever is only part of a much larger problem, that is, the control of infections with hæmolytic streptococci in general. This statement may very well be applied to the control of puerperal sepsis, since hæmolytic streptococci of precisely the same kind as are found in puerperal fever are present in a great variety of commonly occurring septic conditions, such as tonsillitis and scarlet fever, *otitis media* and mastoid diseases, erysipelas, nasal sinus infections and impetigo. As Colebrook[2] has observed, these are almost all open lesions, giving abundant opportunity of dissemination of infection. This list of possible sources of infection is indeed formidable, but it is reassuring to remember that the epidemic sepsis of former times has been eliminated by scrupulous care and the development of aseptic technique. With more care and additional precautions we could eliminate the sporadic sepsis we see today.

The chief danger is probably the throat carrier, for the human throat seems to be the natural habitat of *Streptococcus pyogenes*. It is therefore imperative to examine throat and nose swabs from all midwifery attendants, doctors, nurses and students who develop tonsillitis or other respiratory infection, or who have been in contact with a case of puerperal sepsis.

But it has been estimated that only one-third of the hæmolytic streptococci found in the human throat are pathogenic, so that what is required is a reasonably simple method of distinguishing the pathogenic strains of hæmolytic streptococci from the non-pathogenic strains. The differentiation of hæmolytic streptococci is also important in the matter of cervical swabbings. Leonard Colebrook[2] recommends the immediate recognition of puerperal infection by examining swabbings from the upper part of the vagina or cervix in all cases of puerperal pyrexia and the prompt removal of every infective patient from maternity institutions unless these are provided with a separate isolation block.

The work of Lancefield and Hare[3][4] has shown that the great majority of strains isolated from puerperal infections belong to Group A, the pathogenic group, and that strains isolated from afebrile cases or cases with only slight pyrexia are almost invariably non-pathogenic. Our small series of cases fits in fairly well with this finding. Thirty-one of the forty-eight positive cervical swabbings listed in Dr. Chesterman's paper were grouped serologically by precipitin tests. This was done for us by Professor H. K. Ward in the Bacteriology Department of the University of Sydney. Of these

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

31 swabbings, 27 were shown to belong to Group A. Of the four which did not belong to Group A, one was afebrile and the other three showed only very minor degrees of pyrexia. Without the precipitin test it might be difficult to distinguish such cases from the milder type of Group A infection. It is important to do so in order that one may isolate the patients infected with pathogenic streptococci.

Before discussing this question of differentiation further, it is not out of place to define what we mean by a hæmolytic streptococcus. As Topley and Wilson[5] state in the recent edition of their book, this is by no means always an easy matter. The term hæmolytic streptococcus is usually applied to those strains which produce β hæmolysis on blood agar plates. By β hæmolysis we mean sharply defined, clear, colourless zones of hæmolysis, quite distinct from the greenish coloration produced by *Streptococcus viridans*. The accompanying list taken from Topley and Wilson[5] will serve to refresh your memory as to the way in which streptococci may be classified into three (or possibly four) different categories according to their effect on blood containing media:

Classification of Streptococci.

1. Hæmolytic streptococci: These produce β-hæmolysis on blood agar plates. Two subgroups: (*a*) those that produce a filtrable hæmolysin, (*b*) those that do not produce a filtrable hæmolysin.

2. Streptococci of viridans type. Produce α-hæmolysis on blood agar plates (partial hæmolysis with greenish discoloration).

3. Streptococci that have no action on blood media under the usual conditions of testing.

To the three categories on the list Brown[6] added a fourth variety, producing what he called α' hæmolysis. This term is applied to colonies surrounded by a zone of hæmolysis which is slightly hazy and less sharply limited than is the case with true β hæmolysis.

It may be difficult, unless one is very sure of the media one is using, to distinguish these from true hæmolytic streptococci. The matter of media is important. It is better if possible to use horse blood, or if a supply of this is not available, human blood. Horse blood has been used in most of the standard work on streptococci. The blood of other animals should not be used, as the red corpuscles of different animal species vary widely in their resistance to different hæmolytic agents. We found it difficult at first to obtain a supply of sterile horse blood, and the greater part of our work was done with plates made with human blood.

All swabbings were plated out as soon as possible after being taken and were incubated aerobically for twenty-four hours. The number of colonies of hæmolytic streptococci found in throat swabbings varied; the swabbings with numerous colonies came almost always from persons who had had recent tonsillitis, sore throat or sinusitis. It is interesting to note that in the cervical swabbings hæmolytic streptococci, if present at all, were present in large numbers or in pure culture.

Having then identified the hæmolytic streptococcus, one looks for a method of distinguishing the pathogenic from the non-pathogenic strains. Many workers have attempted to devise such a method. The varying appearances of colonies on blood agar plates are suggestive, but not sufficiently reliable to be of any real use. Taylor and Wright[7] in 1930 were unable to distinguish by any of the tests at their disposal between the organisms isolated from the vagina in febrile and afebrile cases. Animal inoculation is too variable to be of any use, since laboratory animals are resistant to many of the strains which have been isolated from severe human infections. Hare[8] attempted to derive a method of differentiation from the fact that most saprophytic strains can be distinguished from infecting strains in being susceptible to the bactericidal action of human blood. Hare and Colebrook[9] also showed that biochemical tests can be employed to differentiate saprophytic from infecting strains. These tests are hardly suitable for routine laboratory work. Other workers have differentiated hæmolytic streptococci according to their antigenic properties. Lancefield,[13][14] instead of employing the agglutination test, prepared extracts from different strains of hæmolytic streptococci and tested them against antisera by means of the precipitin reaction. She was able to differentiate at least three different antigenic components in her extracts. One of these components is a complex carbohydrate, and it is this substance which gives the precipitin reaction. All hæmolytic streptococci possess this carbohydrate antigen, which Lancefield named the "C Substance", but it varies in structure according to the source from which the streptococci are derived and its variations can thus be used as a means of classification. Table III in Dr. Chesterman's paper published herewith shows this classification into groups. Almost all the strains isolated from human infections possess the same carbohydrate antigen and thus form the group which Lancefield called Group A, the human pathogenic group.[1] When the hæmolytic streptococcus isolated from a throat carrier gives a Group A reaction to the precipitin test, we regard that carrier as potentially dangerous; when a cervical swabbing from a puerperal patient contains a Group A streptococcus, that patient has an infection which is at least potentially dangerous to herself and is certainly dangerous to other patients who are in the same ward or nursed by the same nurse.

The precipitin test is therefore an extremely useful test. Unfortunately it takes a considerable time to perform, more time than is available in a small laboratory where all the routine pathology and biological chemistry of the hospital are done by one medical practitioner and one technician.

As I have just stated, when the hæmolytic streptococcus isolated from a throat carrier gives a Group A precipitin test, we regard that carrier as potentially dangerous. But the precipitin test does not provide a means of identifying a carrier as the source of infection in any particular case; it

[1] See footnote in Dr. Chesterman's paper on page 238.

only shows that such a carrier is a possible source of infection. We should not be contented until we can identify the source of infection in every case of puerperal sepsis, especially when the case forms part of such small epidemics as still occur. This identification of the source of infection is made possible by the existence of Griffith's types.

We have seen that Griffith[1] has differentiated and numbered twenty-seven different types of hæmolytic streptococci by the method of agglutination and agglutinin absorption. Nearly all these type strains, which were isolated from various human infections, belong to Lancefield's Group A. Griffith's work is extremely important; he has, perhaps, done more to increase our knowledge of streptococcal infections than any other worker. His work originated in an attempt to determine the type of streptococcus concerned in various epidemics of scarlet fever. He collected strains not only from cases of scarlet fever, but also from other streptococcal infections. His serological classification first helped to show what had long been suspected on clinical and epidemiological grounds, that a variety of streptococcal illnesses could be coordinated epidemiologically, that is to say, that the same serological type of hæmolytic streptococcus could be responsible for any one of the many varieties of streptococcal infection. Smith[11] and Dora Colebrook,[12] working independently, were able to use the method of serological identification in order to collect evidence on the source of infection in puerperal fever.

For the purpose of identifying the source of infection it is a useful fact that there are at least twenty-seven distinct types of hæmolytic streptococci within Group A. For it means that if we find that the infecting organism in a patient with puerperal sepsis belongs, say, to Griffith's Type 12, and if we find a Type 12 streptococcus in the throat of one of her contacts, or in her own throat, or in the impetiginous sores of one of her children, we have found at least a probable source of infection. Dora Colebrook[12] was able to establish some such probable source of infection in 76% of her 67 cases.

At Queen Charlotte's Hospital every strain of hæmolytic streptococcus isolated from patients is classified by means of Griffith's type series, and also grouped by means of the precipitin test described by Lancefield. This, of course, is possible only in a hospital where the laboratory has a large staff of research workers. This fact needs emphasis because clinicians who are familiar with the typing of pneumococci and who have read of the work of Dora Colebrook will lightheartedly suggest that all streptococcal infections should be typed as they occur.

The carrier problem presents many difficulties. Table I shows the result of swabbing the throats of eighty-five nurses at the Women's Hospital in June, 1937. Streptococcal infections are more prevalent in the colder months. This table probably shows a maximal incidence:

Of the nine nurses whose throat swabbings showed numerous colonies of pathogenic hæmolytic streptococci three had had recent attacks of severe tonsillitis, two had had heavy colds, one had had laryngitis, one developed an acute sinus infection next day, and one had a history suggestive of sinus infection.

TABLE I.

Results of Throat Swabbings of Nurses, June, 1937; 85 Swabbings Taken; Total Positive, 27.

Type.	Number.
Pathogenic	15[1]
Non-Pathogenic	12
Total	27

[1] Of the swabbings yielding pathogenic cocci, nine showed numerous colonies and six few colonies only.

In July, August and September, 1936, swabbings were taken from 104 nurses and resident medical officers within the hospital. At the time we were unable to distinguish between pathogenic and non-pathogenic strains. The results are, however, of interest, in that 14 of the 19 positive swabbings were obtained from nurses who were suffering from severe sore throats or tonsillitis, and who were sent for throat swabbings for that reason.

TABLE II.

Results of Throat Swabbings of Nurses, Resident Medical Officers and Students, July 10 and September 21, 1936.

Observation.	Hæmolytic Streptococci Found.	Hæmolytic Streptococci not Found.
Routine throat swabbings from 86 healthy persons.	5 (6·2%)	81
Throat swabbings from 18 nurses complaining of sore throats, tonsillitis, etc.	14 (77%)	4

Of 19 positive swabbings in the 1936 series, 14 or 74% came from persons who had acute sore throat or tonsillitis. Of nine positive swabbings in the 1937 series, swabbings in which the streptococci were shown to be pathogenic, six came from persons who had had recent infections of the upper respiratory tract, and one came from a nurse who was apparently incubating an acute infection. While it is evident from these two series of swabbings that it is extremely useful to be able to distinguish between the pathogenic and non-pathogenic types of hæmolytic streptococci, it is also evident that the dangerous people are those who have had recent acute sore throats or sinus infection. Where no bacteriological work is done the danger of infection can be fairly well combatted by keeping such people away from the labour ward. In such places, of course, throat swabbings could be sent by post to a central laboratory. The hæmolytic streptococcus is a hardy creature, and Colebrook[2] records the growth of hæmolytic streptococci from swabbings taken forty-eight hours previously and sent to him by post. Those who practise in remote districts may be glad to bear this in mind. A negative result in such a case would not, of course, have the same value as a positive result. The most difficult prob-

lem of all from the point of view of hospital administration is the question what is to be done with the carriers of hæmolytic streptococci. It is impossible to suspend nurses or students from duty indefinitely. It is a serious matter to deplete the nursing staff of a hospital. Each individual is a problem in himself and herself. Acute infection may clear up in time, but it is often a very long time before the throat swabbing becomes free from infective organisms. Some cases clear up after tonsillectomy. Some positive swabbings persist even after tonsillectomy. These may be due to a naso-pharyngeal infection. Nasal sinus disease or infected adenoids may be present. Dental sepsis may be the focus of infection.

The question has been asked whether it is possible to mask carriers so efficiently that they are not dangerous. The masks in common use are usually inefficient and unpleasant affairs. Eight thicknesses of gauze are required to make an efficient mask. The type of mask used at Queen Charlotte's Hospital shaped like a hood is better than the straight pieces of gauze in common use. It seems likely that droplet infection may be conveyed via the gloved hand. If so, the practice of smearing gloves with 30% "Dettol Cream"—a routine practice at Queen Charlotte's Hospital—is to be recommended. Even when these precautions are scrupulously observed, it is, I think, advisable for these persons whose throat swabbings yield numerous colonies of pathogenic hæmolytic streptococci to keep away from the delivery room and to take no part in the after-treatment of the puerperal patient or in the care of the new-born.

One important fact that does not seem to be widely known is the fact that the hæmolytic streptococcus is just as dangerous to the new-born as to the puerperal patient. At the Women's Hospital, Crown Street, Sydney, the neo-natal death rate is not higher than in other institutions, but since we have been on the lookout for infection we have found several neo-natal deaths due to infection with a hæmolytic streptococcus. The diagnosis is not always obvious, and some cases might easily be missed where routine bacteriological examinations and autopsies are not done. The route of infection appears to be sometimes the respiratory tract, as is evidenced by the presence of Group A hæmolytic streptococci in nasal swabbings from infants which subsequently suffered from bronchopneumonia or meningitis. Sometimes the umbilical stump is infected and minor finger infections may also play a part. There appears to be a seasonal incidence. Several cases occurred during the winter of 1936; there were no further cases until the early part of the following winter, when several more infections occurred, some of the patients recovering. Since June, 1937, no further case has occurred.

Acknowledgements.

We hope to publish a full report of these cases later. In conclusion, it is a pleasure to thank Professor H. K. Ward, of the Department of Bacteriology in the University of Sydney, without whose valuable advice and assistance these investigations could not have been undertaken.

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BRAIN ABSCESS.[1]

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EVERY brain abscess is a problem in itself, and one is always face to face with major or minor difficulties either in diagnosis, localization, operative technique or in the all-important after-treatment. The success or failure in the treatment of brain abscess depends on correct surgical judgement of a difficulty peculiar to the particular individual and on the understanding of the pathology, neurodiagnosis and management of problems common to many of these cases.

This paper is based upon the observation and treatment of nine patients and on a study of the available literature. The details of these cases are set out in Table I.

Ætiology.

The ætiology of this condition is well known.

1. Chronic suppurative *otitis media* and chronic mastoiditis in a large series would be the most fre-

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

TABLE I.

Case.	Sex.	Age.	Source of Infection.	Location.	Result.
I	Male.	15	Acute right mastoiditis.	Right temporal lobe abscess.	Recovery.
II	Female.	23	Acute right mastoiditis.	Right temporal lobe abscess.	Death.
III	Male.	67	Subacute exacerbation of right chronic suppurative *otitis media*.	Right cerebellar abscess.	Death.
IV	Male.	—	Left chronic suppurative *otitis media*.	Left cerebellar abscess.	Death.
V	Male.	9	Acute right mastoiditis.	Right cerebellar abscess.	Recovery.
VI	Female.	25	Right pansinusitis and orbital cellulitis, osteomyelitis right frontal bone.	Right frontal lobe abscess.	Recovery.
VII	Male.	21	Left pansinusitis and orbital cellulitis.	Left frontal lobe abscess. Right frontal lobe abscess.	Death.
VIII	Female.	8	Left subacute mastoiditis.	Left temporal lobe abscess.	Death.
IX	Female.	12	Right acute mastoiditis.	Right temporal lobe abscess.	Recovery.

quent cause of cerebral abscess, about 80% of cases arising thus, and especially in those in which cholesteatoma is present.

2. Acute suppurative *otitis media* and acute mastoiditis also give rise to temporal lobe or cerebellar abscess, the cerebral invasion depending on the virulence of the infection, the direction of spread and on the type of mastoid present. This latter factor is extremely important. It is less likely that infection will spread inwards in a highly cellular mastoid, especially if there is only a moderately thin layer of bone between the antrum and cortex. In the acellular type, in which pneumatization has not occurred, or has only partially occurred where the bone is sclerotic and the antrum is deeply situated, invasion of the middle or posterior fossa is far more likely to occur.

3. Frontal sinusitis is the most frequent cause of frontal lobe abscess. It may be acute or chronic, uncomplicated, or part of a pansinusitis; or the frontal sinus may be involved following ethmoidal infection or orbital cellulitis and abscess.

Diagnosis.

It is not proposed to deal at all fully with the diagnosis of brain abscess, but to stress some of the more important points. Many cases successfully treated are not diagnosed until fairly late when the condition is well advanced and localized, and conversely many cases diagnosed early are unsuccessfully treated. The inference is obvious, early diagnosis is not necessarily indicative of early operation. It is, however, important to make the diagnosis as soon as possible so that the progress of the abscess may be followed closely until the most favourable time for operation.

The earliest signs of cerebral abscess are indefinite and none pathognomonic. Individually they may not seem important, but collectively they may suggest cerebral suppuration. Macewan[1] states that invasion of brain tissue by infection is always accompanied by chilliness or a rigor, and often by vomiting, headache and a rise of temperature. These symptoms are due to a cortical encephalitis and last only for a short time. They are those of any acute infective process and it is their association with a discharging ear or a frontal sinusitis or orbital cellulitis that makes us realize their seriousness.

Later the most outstanding symptom is headache, from which the patient is never free, and which varies in severity. Localization is not important and the position may have no relation to the site of the abscess. During this stage of development of the abscess the patient is never well and anorexia, some wasting and pallor are usually present. The patients are often dull, listless and apathetic. Cerebration may be slow. These symptoms, associated with headache and a possible focus, should always arouse a suspicion of cerebral abscess.

The pulse rate is not constantly slow until compression occurs. Early with encephalitis the pulse rate is raised and runs parallel with the temperature, but frequent examinations of the pulse-temperature ratio will show an occasional drop in pulse rate far below the normal for the corresponding temperature. This observation is very important and the ratio may last for only a short time. Frequent readings are therefore essential; temperature may be normal, subnormal or slightly raised.

Convulsions are an important sign and, associated with aural or nasal suppuration, they always mean intracranial extension—a cortical involvement by the inflammation. Cushing states that convulsions are more common with temporal lobe involvement. They are usually generalized and leave a contralateral paresis, partial or complete, which persists for varying periods. Convulsions due to frontal lobe abscess are usually general and often commence with a conjugate deviation of the head and eyes to the opposite side. They are often Jacksonian in type.

Cerebro-Spinal Fluid.

Repeated examinations must be made and careful note kept of results so that changes may be observed. Lumbar puncture is the usual method, but care must be taken as it is not free from risk. One cubic centimetre is all that is necessary for examination and catastrophe may follow the removal of large amounts, for example, medullary corking (especially with cerebellar abscess), medullary hæmorrhage from sudden release of pressure, or rupture of an abscess into the ventricle if it is close to the latter. Subtentorial abscess presents a far greater risk than one above the tentorium, and if suspected the utmost care and precaution must be exercised, the finest of needles being used and a minimum of fluid being removed.

Pressure Changes.—The normal pressure of the cerebro-spinal fluid is 100 to 200 millimetres of

water. Inflammatory changes in the brain and its coverings cause an increase in pressure and rate of flow, and the amount of change would vary with the extent of the inflammation and the amount of œdema. Manometric readings are essential, as it is most unreliable to attempt to estimate early changes of pressure by counting drops per second.

Cell Count.—An estimate of the cell count gives us the greatest amount of information in cases of suspected abscess. Normally the fluid contains one to five mononuclear cells per cubic millimetre. A higher count indicates meningeal involvement, and the greater the area involved the greater will be the cell count. Therefore with involvement of brain tissue and in abscess it can vary from a minimal area of meningitis to a large area involved by superficial or subcortical abscess. In the early stages of purulent encephalitis a greater area would be involved than later on when localization is occurring and the cell count would vary accordingly. In the latent stage of abscess formation when the abscess becomes localized the cell increase may disappear and one may even obtain a clear fluid without an abnormal number of cells. Compare this with a non-abscess meningitis when the cell count is parallel with the severity of the condition. Borries[3] terms the abscess-meningitis readings "benign spinal fluid picture", and states that such a picture coinciding with a malignant picture of other clinical symptoms suggests a secondary meningitis, most often secondary to a cerebral, cerebellar or subdural abscess. If the abscess increases in size and approaches the surface the cell count again rises because of meningeal involvement.

Cell Type.—Polymorphonuclear cells are those most usually found in primary meningitis and mononuclear cells are more common with secondary meningitis. Polymorphonuclear cells are also usually present in the early stages of abscess and with an abscess leaking into the ventricle or subarachnoid space.

Chlorides.—Estimation of the chlorides is the only chemical investigation that is useful, the normal being 725 to 750 milligrammes per 100 cubic centimetres. Readings less than 700 milligrammes indicate a generalized meningitis. Greenfield and Carmichael[2] state the chloride content becomes less as the abscess approaches the surface. If the abscess is deep and encapsuled the chloride value is normal. They also state that a content as low as 680 milligrammes is always fatal, and that the greater the meningeal area involved the greater the fall in chloride.

Bacteriology.—The fluid is usually sterile unless the meninges are affected. Organisms may be present with an abscess leaking into the ventricle.

The importance of repeated cerebro-spinal fluid readings is obvious. In the early stages knowledge of its content is important for diagnosis, and, when the diagnosis is made, one can follow the progress of an abscess to some extent and thus obtain some idea of the optimum time for operation.

Localization of Cerebral Abscess.

Definite signs of localization may not appear until late, and often, especially in the cerebellum, do not appear at all. Cases III and IV were examples of silent cerebellar abscess.[1] Local signs in an abscess depend on the surrounding encephalitis and œdema. An acute abscess formation may have a large area of encephalitis and œdema surrounding it. Hence, there may be many signs, and the central area of infection would be indicated by the early signs. A chronic abscess, firmly encapsuled, has only a small amount of surrounding œdema, and localizing signs are often not present until it is large, and then it resembles a tumour The signs are due to pressure on contiguous areas, such as internal capsule or cortex.

Temporal Lobe Abscess.

The most characteristic sign of a left temporal abscess is aphasia. Typically, it is a paraphasia in which the patient makes mistakes in naming familiar objects. It has been recorded occasionally with an abscess in the right temporal lobe. In more advanced cases the loss of memory for word sounds may be very pronounced, so that words and syllables are mixed and speech becomes almost unintelligible.

Paralysis is the most constant sign and consists of a contralateral paresis. This is due to involvement by pressure or œdema of the motor cortex, *corona radiata* or internal capsule. The face area is nearest to the temporal lobe with arm, leg and foot higher on the cortex. Hence face and arm are first affected. Symonds[4] states that the most important sign of temporal lobe abscess is slight weakness of the opposite side of the face in its lower half, with symptoms of pyramidal tract involvement. Occasionally the only sign of the latter may be an absent abdominal reflex. Third nerve involvement on the same side is often present. The oculo-motor nerve is closely related to the temporal lobe on the side of the body of the sphenoid and extension and pressure downwards towards the floor of the skull will cause dilatation of the homolateral pupil, followed by ptosis, and later paralysis of the external muscles supplied by the third nerve.

Jacksonian epileptic attacks may occur and vary with the site of the encephalitis and œdema. Repeated examination of the visual fields must be made, and the development of a quadrantic hemianopia would make the diagnosis certain. Usually it is an upper quadrantic hemianopia, as the abscess is low in the temporal lobe, but occasionally it localizes in a higher position, involves the upper fibres of the optic radiation and produces a lower quadrantic hemianopia. Important lateralizing signs are pain in the teeth and pain behind the eye from pressure on the Gasserian ganglion.

The diagnosis of temporal lobe abscess, while often obvious, may be extremely difficult. If a patient who has a discharging ear or who has had an aural operation develops aphasia, a contralateral paresis or other symptoms suggestive of

[1] Dr. Cantor had prepared and circulated amongst his audience a short *résumé* of the clinical histories of the patients in his series.

intracranial suppuration, the diagnosis is easy. But given only a lost lower abdominal reflex in the presence of a discharging ear and a headache it may be extremely difficult, and a careful day to day examination must be made, together with a summation of all the available evidence.

Frontal Lobe Abscess.

Cases VI and VII in this series are examples of frontal lobe abscess, Case VI arising from osteomyelitis of the frontal bone and Case VII following frontal sinus suppuration. The diagnosis of abscess in this area is notoriously difficult, as it is a silent area and usually depends on the presence of infection in an area known to be dangerous, associated with symptoms suggestive of intracranial extension. The dangerous foci are frontal sinusitis, suppurative ethmoiditis and orbital cellulitis, and osteomyelitis of the frontal bone. Diagnosis may be made at operation by discovery of bony necrosis on the posterior wall of the frontal sinus. Frontal headache, exacerbation of an old latent infection associated with papillitis, vomiting, low pulse–temperature ratio, or indeed any signs of cerebral suppuration, is strongly indicative of frontal lobe abscess and justifies exploration. The most constant localizing sign, as pointed out by Watkins,[5] is absence of the homolateral abdominal reflex. Contralateral paralysis or paresis may occur, and Eagleton[6] states that sudden onset of paralysis in the contralateral arm is pathognomic. Both these signs were present in Case VII.

Cerebellar Abscess.

Diagnosis of cerebellar abscess may also be easy or difficult. Abscess of the cerebellum arises secondary to lateral sinus thrombosis, perisinus abscess or labyrinthitis, and the detection of cerebellar symptoms, associated with any of the general signs of intracranial suppuration and the presence of any of the above conditions, makes diagnosis reasonably certain. The difficulty arises in those cases which are neurologically silent, and there are many of these. The majority of cerebellar abscesses start in the cortex and do not spread beyond it. According to Gordon Holmes, localizing signs are due to involvement of the dentate nucleus. Hence an abscess in the cortex, far removed from the dentate nucleus and with little or no surrounding œdema, would show no characteristic signs.

In diagnosis of "silent abscess" one has to rely on the history and on the symptoms and signs common to cerebral suppuration and intracranial pressure. Headache, vomiting and papillœdema are more intense with subtentorial abscess than with abscess above the tentorium. Also other signs common to a lesion in the posterior fossa may be seen, such as neck stiffness, suboccipital tenderness and a positive Kernig's sign. Cerebro-spinal fluid examination is important, but the proceeding is very dangerous and the amount withdrawn must be minimal.

If localizing signs are present they are often transient, as they depend on œdema. Rarely are all the classical signs of tumour present, and the commonest are nystagmus, a pointing error, hypotonia and dysdiadockokinesia.

Treatment of Cerebral Abscess.

Probably in the whole realm of surgery there is no more controversial subject than the treatment of cerebral abscess. Every possible aspect is the subject of argument and discussion; surgical approach, methods of exploration, methods of drainage, treatment of the cavity, length of drainage, indeed, every step in treatment has its advocates and antagonists.

As previously mentioned, early diagnosis does not necessarily indicate early operation. One has only to think in terms of pathology to realize how futile it is to attempt to drain an area of acute suppurative encephalitis. If the inflammatory process limits itself and becomes walled off, complete softening and disintegration of the localized area occurs, and drainage is then possible. Also the subarachnoid space is sealed off and there is less risk of leakage. Early treatment of the causal condition is imperative, for example, a suppurative frontal sinusitis or a chronic mastoiditis, but the decision to explore the brain should be withheld until one is reasonably certain by cerebro-spinal fluid findings and clinical examination that localization has occurred. In practice this is often difficult to do, and one feels forced to operate, but results are far better in subacute or chronic cases. Also, there are many cases which never become localized, but are progressive. These are always fatal. Rarely is operation for brain abscess urgent. If failure of respiration occurs with cerebellar abscess rapid decompression is necessary. When a patient becomes unconscious because of rupture of an abscess into the ventricle, or, as occasionally occurs, with a marginal temporo-sphenoidal abscess, operation is also urgent, although there is always time for exposure. In practically all other cases the operation time is chosen.

Approach to an Abscess.

Temporo-Sphenoidal Abscess.—If the suspected abscess follows an acutely inflamed ear simple mastoidectomy is sufficient to give good exposure of the *tegmen antri*, but if chronic middle ear suppuration is present the radical operation is performed to expose the *tegmen tympani* as well. The plastic part of the operation should be left until the abscess has been successfully treated. The tegmen is carefully examined for fistula or any area of necrosis, and if this cannot be found the dura is widely exposed and examined for discoloration or lack of pulsation. If none is found and it is decided to explore the brain, further exposure should be made by removing part of the squama. Some authorities favour exploration via the temporal route because exploration is made through a clean area. If, however, an abscess is found, there must be greater risk of infecting the subarachnoid space which is not at this site sealed by adhesions. Generally speaking, the mastoid approach is most favoured for exploration and drainage is along a preformed tract.

Frontal Lobe Abscess.—The majority of frontal lobe abscesses originate in frontal or ethmoidal infection and the abscess is best reached via the frontal sinus. The posterior plate and dura are carefully examined for evidence of a track. Frontal lobe abscess is very difficult to drain, and this is one of the main reasons for the high mortality. It must be almost impossible to obtain good access and drainage from the infero-medial aspect of the frontal lobe, as any tube through the sinus is at an awkward angle. If exploration via the frontal sinus reveals an abscess high up in the frontal lobe or otherwise awkwardly placed for drainage, direct external approach should be made.

Cerebellar Abscess.—It is probably better to follow the tract of infection if possible in cerebellar abscesses, as it is in temporal and frontal lobe abscesses. The majority of cerebellar abscesses are located in the anterior-inferior part of the cerebellum. In cases of sinus thrombosis the tract is through the sinus. This should be obliterated and the cerebellar dura exposed through it. If no such change is present, or if the abscess is of labyrinthine origin, an attempt should be made to expose the dura in front of the sinus. Ease of access will depend on how far forward the sinus is. Exploration should be made forwards and inwards and directly inwards up to about 3·0 centimetres (one and a quarter inches). If exploration is unsuccessful, exposure should be made behind the sinus by a horizontal incision towards the occiput. The bone is trephined 2·5 centimetres (one inch) behind the bend and below the level of the lateral sinus. Exploration should be made inwards and forwards up to one inch, inwards and slightly forwards up to 3·75 centimetres (one and a half inches), and directly inwards up to 3·0 centimetres (one and a quarter inches). (Kerrison.)

During exposure special attention is paid to any areas of bony necrosis. Dura is carefully examined for discoloration or granulations, and occasionally a bead of pus is seen exuding. Normal dura, provided there is clinical suspicion of abscess, in no way contraindicates exploration. It is not at all uncommon to have normal dura in the presence of an abscess.

Exploration.

It is safer for exploration to use blunt instruments, such as a wide bore, blunt exploring needle or blunt aural forceps, rather than a sharp scalpel, as the last mentioned may easily puncture an intracerebral vessel and cause serious or even fatal hæmorrhage. Blunt forceps can also be slightly opened and pus which would not flow down a canula can pass down the tract. The whole field of operation should be cleaned before the dura is opened, towels and gloves should be changed and fresh instruments used. A small incision should be made in the dura and an exploration made through it. If exploration is unsuccessful, the exploring instrument is completely withdrawn, a fresh dural incision is made and an exploration is made in a different direction. For exploration Kerrison[7] advocates passing the instrument through small parallel dural incisions. His reasons for reexploration through fresh incisions are as follows:

1. There is no danger in a clean, small, dural incision.
2. If pus is not found at first and later bursts through and drains along one puncture tract when a number of explorations have been made through the same opening the surgeon is left in doubt as to the site of the abscess.
3. If pus is evacuated after several punctures through the same opening the infection may pass up the previous tracts and cause fresh foci.

These are extremely important practical points.

Drainage.

There are many methods of drainage, and the one used should depend on the position and accessibility of the abscess. Whichever method is used it must be simple and no force should be used in its application. The utmost gentleness is necessary in applying drainage to an extremely delicate and inflamed tissue. Single tubes, double tubes, silver tracheotomy tube, cigarette gauze wick rolled in iodine and boric powder, Mosher copper wire drain are amongst the many materials advocated. The difficulty with a single tube is the fact that it may become blocked, and once it is removed it may be impossible to replace it without damage to the delicate surrounding tissue. If a single tube is used it should be left in until all drainage ceases and it should be gradually shortened over a fairly long period. Le Maitre's method may be useful when one cannot get dependent drainage, as in the frontal or cerebellar area. It consists in making a small opening in the dura large enough for a tube the size of a Number I catheter to be passed along the forceps into the cavity. Every other day the tube is replaced by a slightly larger one. The difficulty, of course, is that it may be impossible to replace a tube once removed without damage to inflamed brain tissue. Probably one of the best methods is the two-tube method advocated by Atkinson.[8] One tube of not more than six millimetres (a quarter of an inch) bore and the other twice this size are placed alongside each other. They are not joined and reach just into the abscess cavity. They are stitched to the integument. The smaller tube is for irrigation, saline solution being run into the cavity during after-treatment, and the larger is an outlet for saline solution and a drain. The smaller tube is used as a guide if the other needs to be changed.

Post-Operative Treatment.

Successful drainage is indicated by an improvement in the general condition of the patient. One of the most outstanding features is the complete relief of headache. If the patient responds to drainage the immediate treatment is to do as little as possible. If one tube is used, on no account should it be removed for at least a week till a well-formed track is present. The difficulty of replacing a tube removed before the track is well formed is apparent only to those who have had the management of these cases. Forcible replacement may cause a fatal spreading encephalitis, a meningo-encephalitis, or intracerebral spread, with fatal secondary abscess. The outer dressing may be changed as

often as necessary and must not press on the tube. The main dressing, that is, attention to the tube, should always be done by the surgeon himself. A single tube may be carefully mopped out or gently turned if drainage is not quite satisfactory or if the pus is thick. After a week it can be removed for cleaning, and, as drainage lessens, smaller tubes can be used.

After an early marked improvement there may be a return of headache, apathy, drowsiness and vomiting. This is probably due to unsatisfactory drainage or to the formation of a second abscess. The patient should be taken to the theatre, as it is impossible to examine him satisfactorily in bed. No anæsthetic is necessary. The most common cause of imperfect drainage is a bad position of the tube. If it is placed too far in, the roof of the abscess tends to contract down and block it, or the tube may be too small for the passage of thick pus. A little manipulation may free retained pus which wells out of the cavity.

More difficult to discover is a second abscess in a pocket. If the wall of the abscess is injured by the tube inflammation may occur through it and fresh foci form. If manipulation of the tube or inspection of the cavity fails to reveal the focus, the surrounding area must be explored and any other collection treated as the original abscess. Encephalitis in the area surrounding the abscess or spreading beyond this, recurrence of localized œdema due to encephalitis, or meningitis due to leakage into the subarachnoid space, may all retard recovery or may be fatal. Encephalitis may follow too frequent tube manipulation or imperfect drainage. Focal symptoms or fits may occur, as in Case VI, and cause considerable anxiety as to whether a second abscess is present. A most important observation is the pulse–temperature ratio, and in the absence of pocketing or retention the pulse is usually rapid and the temperature raised. Frequent lumbar puncture and intravenous administration of hypertonic saline solution will often control the condition.

The commonest cause of death is meningitis. This may occur if the subarachnoid space is not sealed off by adhesions, if the abscess is not well localized, or if drainage is not complete. Occasionally one may get a combination—a spreading meningo-encephalitis with numerous and changing focal symptoms, fits, signs of meningitis with turbid spinal fluid, and a raised temperature and pulse rate. Quite often this condition is brought about by too early operative interference before the abscess has localized and is usually fatal.

After Results.

The future health of patients who have recovered from an abscess of the brain depends on how much cerebral tissue has been destroyed by the abscess and operation. Also important is the position of the abscess. Recovery from cerebellar abscess is usually complete. It is probable that only in cases of a small abscess near the surface is intellectual recovery complete, and, if much damage is done during after-treatment, the advantage of an abscess of this type may be lost. Recurrence of infection may occur in an abscess cavity not fully obliterated and this may occur years after the original infection. Epileptiform seizures due to cortical fibrosis or adherent dura may occur, headaches are common, and especially with frontal lobe abscess changes in mentality. A three-year period at least should elapse before one could feel reasonably certain that fresh foci will not form.

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PSYCHIATRY OF THE SCHOOL PERIOD.[1]

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Mental Deficiency.

I PROPOSE to pass over the large and important subject of mental deficiency, since it was dealt with at length at the first congress in Melbourne in 1924. Diagnosis usually presents no great difficulty, and in primary cases the treatment consists in providing training suited to the child's capacity and designed to afford him a maximum of happiness and independence in later life and perhaps even render him capable of being self-supporting.

Dulness and Backwardness.

On this side of the ill-defined and vaguely delimited frontiers of mental deficiency, we find a wide terrain full of confusion, comprising the so-called submerged tenth, which includes what has been called the "social problem group" (Norwood East), the "dull and backward group" (Burt) or briefly labelled the class of "C" children. Much confusion of terminology exists; and this requires some discussion. In mental hospital practice it has long been the custom to describe an individual as "dull" in whom an affective atrophy has taken place, as in the end stages of schizophrenia, but to describe him as weak-minded, when the defect is predominantly intellectual. On the other hand, the use of the term "dull" to describe a child who is lacking in intelligence is just as firmly established with teachers; and educationists and psychologists generally follow this practice. Both usages are so firmly rooted in precedent that it will be difficult to find acceptable synonyms; yet scientific precision is jeopardized by an ambiguity of terms. It is common, for instance, to hear a mental defective described as "bright" or "dull" according as he exhibits lively interests and adequate emotional responses in relation to his general mental development or the reverse.

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

Following this usage, one would be led to speak of "bright" or "dull" dullards. Without attempting a solution, we merely state that in what follows "dulness", unless qualified, will be used to designate a condition of poverty of intelligence not amounting to mental deficiency.

"Backward" and its latinized synonym "retarded" are also used loosely—often when it is desired to designate a "dullard" or even a mental defective. But according to the terminology adopted here, "retarded" will be reserved to designate a child whose actual development has lagged conspicuously behind his innate potentialities and the differential diagnosis between "retardation", and pure "dulness" will be held to be of crucial importance. It will be conceded that, if the experiment were made of rearing a child in an environment of complete isolation from his fellows, protected from all external stimuli such as those of sight and hearing, he would show very little mental development, whatever his innate potentialities. Thus Charlotte Bühler has shown[1] that, on account of a national custom of restricting their movements, the early development of Albanian infants is greatly retarded, but that the lost ground is very rapidly made up when the restrictions are removed. According to our conception retardation occurs alike in mental defectives, dullards, average, superior and gifted children. Unlike mental deficiency and dulness, for which it is often mistaken, it is readily amenable to treatment. Their extremely poor showing at school mentioned in the biographies of many celebrated persons demonstrates that it is frequently overcome, and may give ground for the belief that retardation will right itself, and therefore needs no emphasis. On the other hand, studies of delinquents and criminals show that a very high percentage of these is recruited from the retarded group, and indicate that adequate handling of the problem of scholastic retardation should prove a most important prophylactic measure against these deviations.

In practice, retardation is often relative rather than general. Thus the country lad is likely to be retarded in regard to school work and the social amenities, but to leave his city cousin far behind in his ready self-help and practical manipulation of nature. Burt has made similar comparisons between the slum urchin and the nicely brought-up child of the residential suburb.

Dulness will be distinguished from retardation largely by the results of intelligence tests, but these should always be taken in conjunction with a careful history taking and physical examination. The important considerations are that the dullard's vocational horizon is likely to be limited to unskilled labouring or artisans' work and that his school curriculum should be adapted accordingly. While teachers should be content with slow progress, the emphasis should fall on manual and technical training.

Neuroses and Behaviour Disorders.

In childhood the neuroses are not as clearly delimited as in adults, and are almost always associated with various disorders of behaviour, so that it is convenient to consider these groups together and to use the inclusive term "nervous child" to describe the subject.

We shall first discuss McDougall's conception of primitive passive sympathy.[2] This is the process whereby an emotion is communicated from one individual to another, independently of any of the ordinary excitants of the emotion in the individual, who is sympathetically affected. It is a characteristic reaction of all gregarious species and is the motive of mass reactions as in stampedes, panics, frenzied applause and mob violence. Biological necessity demands that the very young should be especially prone to share the emotional reactions of the older and more experienced members of the group, so much so that infants are more affected by the marks of alarm or anxiety in their parents than by the presence of actual danger.

From the point of view of this discussion the importance of passive sympathy arises from the fact that a guardian's emotions are apt to be induced sympathetically in a small ward and give rise to disturbances which may be difficult to explain, unless careful inquiries are made to elicit facts of this order. The nursling who perceives a tenseness of his mother's muscles when she becomes a prey to some baseless fear regarding him cannot fail to be the subject of the same emotion. If the child is offered nourishment with the apprehension that he will refuse it, sympathetic fear displaces appetite. He is held out to stool with anxious hands and all desire vanishes. Nursing mothers who have been overtaken by some grief or disaster are wont to say that their milk has been "turned", so that the infant will no longer accept it. I have no doubt that most cases of this nature are to be explained by the mother's troubled feelings sympathetically inducing the same emotions in the infant to the exclusion of appetite.

Indeed this emotional language is almost the only language in the lower animals. We have less difficulty in reading the feelings of that most sociable of beasts, the dog, than those of our own fellows, when we see him licking his chops or snarling, with hair abristle in anger or on end in fear, with tail flamboyant or in retreat between his legs. For the infant, too, who is still recapitulating ancestral experiences, this is the only language and he is immeasurably more sensitive to it than are his elders. At this stage, when his cognitive processes are still so inchoate that James saw fit to describe them as "a blooming buzzing confusion", his affective life has already assumed considerable differentiation. It thus comes about that his emotions are almost as dependent on his mother for nutriment as is his body for more material food. Even the toddler will approach without fear a threatening animal or other danger, until his mother has communicated her alarm. A practical point which emerges may be mentioned in passing, namely, that in defending her child from playing with fire, the mother will do well to communicate her fear to

him, so that he feels it is dangerous rather than merely taboo. In the former case she has helped him to make an important step in adaptation to life, while in the latter he is likely to nurse a guilty curiosity until he can satisfy it at a most inopportune moment.

Yet the fretful complaining widow with whom the past is ever present cannot understand why her boy or girl is listless, depressed and has no appetite. In regard to appetite especially, a good example will usually succeed, when precept only defeats its own end. The child who has resisted all the coaxings with tempting dainties in the privacy of the nursery, will partake heartily of the board of hungry convivants. For the same reason it is unwise to display anger when dealing with faulty behaviour or outbursts of temper. The parent should realize that it is only social behaviour on his child's part to smile when he smiles; to laugh when he laughs; to frown when he frowns; and to tremble when he trembles.

In the next place, we may notice that the child has a less stable nervous system and is lacking in the control of the adult. Any emotional reaction is likely to be expressed in marked visceral disturbances. A child may be sent home from school for a vomiting attack, the result of a painful episode in class of an hour ago, to which the teacher attached so little importance as not in any way to associate it with the present disturbance. Next, there appears to be a strong tendency both in children and adults to the formation of patterns both in the reception of stimuli and in discharge phenomena. The mother of the vomiting child may become anxious that he is developing appendicitis and by her questioning and emphasis cause him to pay morbid attention to his visceral sensations. If this is continued for any considerable period, the threshold for such sensations becomes lowered by a process which I have called "the method of special training".[4] At the same time we find that emotional excitement in general tends more and more to adopt one particular mode of visceral discharge—vomiting in the case we are supposing. This is due to the fact that it has served to provide an escape from an unpleasant situation or promoted some other conative satisfaction, by which means it becomes "stamped-in by the method of trial and error". We have now arrived at the stage of adaptation by illness. But it is a mistake to suppose that the motives of all such morbid manifestations are egotistical. Common motives are fear or a painful feeling of inferiority, as usually occurs in stammering and enuresis. On other occasions I have felt that the mother has unwittingly exerted a hypnotic influence on her child. For instance, the subject of habit spasm may show no sign in the *tête-à-tête* interview, but perform antics as soon as he meets the searching glance of his mother, reminiscent of the antics of the showman's subject.

All this goes to stress the very great dependence of the child on his parents. Not only may he be affected in the subtle ways mentioned above, but also in more material ways since he is at his parents' mercy in regard to food, clothing, shelter and so on. He is sure to be the victim of whatever dietary or other fads his mother may have. One condition recurs sufficiently frequently and remains sufficiently true to type to embolden me to give it a special title, which I propose to call the "syndrome of the costive guardian".

The subject is in the custody of an elderly person, more often grandmother than mother. The guardian, who usually suffers from some degree of *prolapsus uteri*, has difficulty in evacuating solid fæces and constantly resorts to the aid of aperients. On mixing her own draught it is convenient also to mix one for her small charge. Projecting her own infirmities on to him, as these old ladies love to do, she also plies him with "All-Bran", prunes and other articles of diet, which create large and irritating residues.

Innocent and necessary as aperients may appear, it is still true that they may be the vehicle of the most crippling and soul-destroying tyranny. Sharp colicky pains may cause the child to double himself up; the symptoms described in vague terms and with emotional emphasis may cause him to be placed in hospital under observation for renal colic or appendicitis. On another occasion tenesmus may produce faintness and collapse, which can easily be mistaken for *petit mal* or a cardiac attack. With this array of symptoms a fertile field is offered for the guardian in which to sow her morbid suggestions, inspired by the pessimism of her years and the dejection of her own infirmities. The patient is likely to lose all initiative and self-reliance, falling entirely under the sway of his tormentor, who now begins to experiment with fresh dietary fads. In a few cases in predisposed subjects it has seemed to me that such a train of events has ushered in a schizophrenic breakdown during the adolescent period.

Delinquency.

In order to study faulty conduct we need to systematize our ideas regarding the development of moral sentiments and character—to study the springs of action and how they become harmoniously arranged and bound together to issue in right conduct. In what follows I propose to adopt McDougall's teaching.[3] Space will not permit me to do more than briefly recall to you his conception of the development of the sentiment as the organization of a constellation of connative dispositions in regard to a certain object: the sentiments, at first crude and poorly organized, becoming more and more complex and integrated, at first particular and concrete only and later becoming general and abstract; the gradual growth of the self-regarding sentiment from the first glimmerings of consciousness to the fullest self-realization; the organization of all the other sentiments within the self-regarding sentiment whence issues volition as the self-regarding sentiment in action in contradistinction to mere impulsive action.

It is obvious that the growth of the sentiments and the development of the character must depend on environment; it is all acquired mental structure. But it is also true that even in the same environ-

ment there are vast individual differences in capacity to acquire sentiments, and this capacity by no means runs parallel with intelligence. To take the extreme example, the constitutional psychopathic inferior or moral imbecile is innately incapable of forming any but the crudest sentiments, so that his conduct cannot be calculated but depends from day to day on the impulse of the moment—there is no *Leitmotif* running through his life. At the other extreme, in "Oliver Twist" Dickens has imagined a lad growing up in sordid surroundings who receives an education in burglary in a highly expert school and yet fails to absorb any of this education, but, on the contrary, in some mysterious way develops principles of probity and honour. Is this a monstrous and impossible fiction, or has the poet's creative intuition seized some of the lineaments of real life? I believe that the latter alternative is the correct one, and the author makes his story more probable in the end by disclosing that his hero was the fruit of the unfortunate love of two high-minded individuals.

Creighton Miller[5] has said that scientific medicine can only accept a deity whose essential function it is to maintain a trustworthy universe. So also is the mark of wise parents, who stand to the infant *in loco dei*, to provide their child with a stable world. How can his character develop where all is caprice; where what is law with one parent is licence with the other; in a world to be cajoled, defied, propitiated or used?

Since solitary creatures require no laws except to provide for their own preservation and the continuance of their species, it is reasonable to suppose that moral sentiments grow out of man's gregariousness. It follows that it is necessary to provide the growing child with companions of more or less his own age. If his associates are adults, on account of his dependence and immaturity he cannot meet them on an equal footing; and this is not favourable to the growth of character. Let us suppose that it was possible to take a dozen boys who had never seen marbles before and to supply a bagful each and an area of level courtyard without any instructions at all. In a few hours we should find them playing industriously with a very workable set of rules vociferously enforced. We have here already the germ of all the codes of man.

There are two conditions in modern society that are rendering the upbringing of children increasingly difficult. These are the tendency to small families and the growth of flat life. The first deprives the child of today of brothers and sisters with whom he may associate as companions on equal terms; and the second deprives him of playing areas where he may meet his fellows. The problem of the only child is far greater than that of dealing with the coddling propensities of insufficiently occupied parents; for the very wisest parents cannot with the exercise of the greatest foresight provide this give and take of the large family. To meet the deficiencies of our present-day social structure, the provision of kindergartens, organized playgrounds, boys' and girls' clubs, boy scout and girl guide troops are advocated. Most important of all, a universal, sound and adaptable educational system is required today as it was never required before.

Mere submissiveness should not be mistaken for steadfastness of character. The seemingly fine character built on precept and religious instruction alone is a hothouse plant which will surely wilt in the face of the first blasts of real life. Let the teacher beware, too, that his favourite pupil is not mirroring too slavishly his own mind and displaying already the seeds of automatic obedience and catalepsy of a future schizophrenic psychosis.

In the case of mental defectives and dullards we cannot look for the highest type of character development. In any case they must always be more or less dependent on stronger minds. Their sentiments are likely to be more concrete in nature; but the self-regarding sentiment, while simple in structure, may still be very stable and well founded. In training them we must rely more on precept and religious sanction and look less for a natural growth of character; but natural growth also takes place in their attachment to persons and places. What their sentiments lack in complexity they may even gain in strength.

Using the word environment in its widest sense as meaning all those influences which operate on the child, it may be said that the moral environment is vastly more important than the material environment—in other words, that his parents' temperamental assets or deficiencies will influence his character development much more than their wealth or poverty. The grandmother often makes an excellent guardian for a young child, but she often fails with the approach of the pubertal period, especially in the case of the bright child who is beginning to feel capable of greater independence and wider interests. To say that with advancing age she is likely to become irritable, weak, vacillating, arbitrary and obstinate is not to reproach her, but merely to mention the characteristics of her years. In such circumstances, so far from learning that honesty is the best policy, her ward may find that prevarication is almost essential to existence. A less subtly minded child may adopt a policy of more or less open rebellion—a sort of reprisal against hampering restrictions and frustrations. Or, again, the less energetic types adapt themselves by illness, which happens very readily, since these old ladies have an inveterate tendency to project their infirmities onto their wards and to apply amateur remedies. In practice there will usually be an admixture of the three types of reaction. And what has been said of grandmothers applies also when the parents are greatly inferior in intelligence to their child.

In dealing with delinquents one obtains a history with remarkable frequency that one or other parent is a pensioner or beneficiary under the *Workers' Compensation Act*. Those cases in which there is room for differences of opinion as to the beneficiary's real capacity seem to have the most malignant influence on home life. This is understandable when we consider that the recipient, living in con-

stant fear of losing his benefit and being prevented from augmenting his limited means except fraudulently, develops a paranoid attitude towards society. He also feels obliged continually to exaggerate his ills, conduct which his children can easily learn to imitate.

Conclusion.

In now concluding this paper I wish freely to admit its lopsidedness and to apologize for having introduced a subject too general for treatment in the time at my disposal. When I came to put together my materials I soon realized that they covered such a wide range that to attempt anything like a balanced treatment would result in little more than a catalogue. I may excuse the omission of any mention of the epilepsies, the encephalitides or cerebral vascular lesions on the ground that they are listed for discussion separately. "The sequelæ of cerebral trauma" might well be chosen as a subject for a later congress, and "speech defects" would be suitable for discussion at a combined meeting. I will leave it to subsequent speakers to point out the many other gaps and count it a virtue to have left them plenty of scope for discussion.

Acknowledgements.

I desire to express my thanks to Mr. G. Ross Thomas, Director of the Department of Education of New South Wales, for permission to use the records of the departmental Child Guidance Clinic for the purpose of preparing this paper; and to Dr. A. E. Machin, Principal Medical Officer, for valuable help and criticism.

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(3) William McDougall: *Loco citato*, page 120 *et sequentes*.
(4) H. M. North: "The Emotions and Functional Disorders of the Viscera", THE MEDICAL JOURNAL OF AUSTRALIA, April 24, 1937, page 624.
(5) Report of Fourth Biennial Conference on Mental Health, *The British Medical Journal*, February 1, 1936, page 226.

INVERSION OF THE UTERUS FOLLOWING CHILD-BIRTH.[1]

By PERCY G. BRETT, M.B., B.S., F.R.A.C.S.,
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THERE are two reasons for my bringing before you such a comparatively rare condition as inversion of the uterus following child-birth. The first is that it has been our fortune or misfortune to have a series of seven cases in the last five years at the Women's Hospital, Melbourne. Three of these patients I have had under my care, and I had an opportunity, which is fortunately not granted to many, to study these cases. I am quite aware that seven cases are not a large series; but when one considers the rarity of the condition it is a comparatively large number. De Lee[1] gives the complication as occurring once in 750,000 cases; Jellett and Madell give it as occurring once in 190,000 cases; Davis,[2] at the Methodist Hospital, New York, gives it as occurring once in 6,500 cases; and at the Women's Hospital, Melbourne, it occurred approximately once in 4,300 cases during the last five years. Added to our series I was able to collect from recent literature a series of five cases reported in the *American Journal of Obstetrics and Gynecology* by D. N. Barrows,[3] of New York, and a series of four cases reported in the same journal by G. H. Davis, of Brooklyn, New York. This makes sixteen cases in all.

The second and main reason for my choosing this subject is that from a study of our cases at the Women's Hospital, and also of those of Barrows[3] and of Davis,[2] I wish to emphasize a line of treatment which I hope to convince you has given better results than that recommended by a majority of the leading textbooks. I am further encouraged to do this by finding that these other observers had arrived at practically similar conclusions as regards treatment after their series of cases.

As I wish particularly to refer to treatment, I shall only very briefly refer to ætiology, to symptoms and to diagnosis.

Ætiology.

Various theories have been advanced as to causation. These may be summarized as follows:

1. Too vigorous massaging of the relaxed uterus, with pressure of the fingers, may indent the fundus and thus start inversion.

2. The routine use of pituitrin or other oxytocic drugs is mentioned by Davis[2] as a possible cause; causing violent contractions of the fundus, the lower uterine segment being relaxed. This may be associated with an inherent flabbiness of the lower uterine segment; it seems likely that if this was not so, more cases would occur, since pituitrin, "Ernutin" *et cetera*, are used almost as a routine measure. Davis says some uteri undergo inversion through any opening, and reports a case in which inversion through the uterine scar occurred during a delay in the operation after an injection of pituitrin. He also states that some patients may be more susceptible than others to the action of pituitrin.

3. Traction on the cord with the placenta attached to the fundus may cause inversion of the uterus.

4. Manual removal of the adherent placenta, if the placenta is dragged on before being completely separated, may drag the fundus down and occasion an inversion.

5. A submucous fibroid tumour may start an inversion.

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

Diagnosis.

The diagnosis is not difficult, provided a careful examination is made; and inversion should be looked for and suspected in all cases in which there are symptoms of grave shock out of all proportion to blood loss. Polypi and a large, bulbous, œdematous cervix may resemble on cursory examination the uterine fundus. Inversion must also be diagnosed from other conditions which cause shock, such as rupture of the uterus and *post partum* hæmorrhage.

Symptoms.

The symptoms may be summed up as intense sudden shock, out of all proportion to blood loss, hæmorrhage often accompanying the inversion (not a marked feature in our cases). The finding of the inverted fundus in the vagina, with the placental site visible or with the placenta attached.

It should be easy to control any hæmorrhage, as the bleeding surface is visible when a speculum is inserted, and packing around the inverted organ can be done thoroughly. The inversion of the uterus must cause some kinking of the uterine vessels, which I think accounts for the fact that hæmorrhage is not such a frequent and serious complication as would be expected.

Complications.

Sepsis is the main complication referred to, and is held up as a great danger in these cases. I should like to draw your attention to the fact that sepsis has never been a cause of death in any of the cases in these series, a mild sapræmia being all that has been noticed. Even in one of my cases, in which the completely prolapsed and inverted uterus fell into the bed-pan, no serious sepsis eventuated. Of course, in all cases aseptic and antiseptic precautions had been taken to prevent infection.

Treatment.

My chief reason in reading this paper is to try to convince you, by reference to our cases at the Women's hospital and to the cases of Davis and of Barrows, that the right treatment, and the one that gives by far the best results, is that of treating the patient for shock and hæmorrhage and of ignoring the inversion for the time being, except for aseptic and antiseptic treatment of the inverted organ. The inversion can be dealt with at any suitable time after the patient has regained her strength.

The operation of choice is by the abdominal route, aided by pressure from the vagina.

The history of our seven cases is summarized in Table I.

Attention is drawn to the following facts: (i) the age of the patients; (ii) the time of recognition of the inversion; (iii) the recovery of all patients in these series in whom inversion of the uterus was not recognized early and who were not subjected to attempts at reduction under general anæsthesia at the time of inversion, with the exception of two cases that were recognized immediately when the placenta was being manually removed, and in which the inversion was partial; those patients who were given a general anæsthetic and subjected to a determined attempt to reduce the inversion, died; (iv) the occurrence of reinversion without any operation in three cases of the series.

TABLE I.

Cases Occurring in and Admitted to the Women's Hospital, Melbourne, 1932–1936.

Patient.	History.	Treatment.	Result.
Aged 30. *2 para.*	Diagnosed thirteen days after delivery. Went out at own risk.	Readmitted three days later. Spinelli's operation.	Recovery.
Aged 18. *Primipara.*	Forceps. Baby 5½ lbs. Six hours later, shock. Inverted uterus found, completely prolapsed.	Gas and oxygen anæsthesia. Vagina packed. Transfusion. Repeated four days later.	Spontaneous reinversion seven weeks later. Two years later, well.
Aged 32. *Primipara.*	Premature baby 5½ lbs. End of third stage inverted uterus found and replaced. Seventeen days later, severe shock. Uterus inverted.	Vagina packed. Transfusion given.	Seven weeks later uterus spontaneously reinverted.
Aged 30. *2 para.*	Normal labour. Baby 7½ lbs. Mild *post partum* hæmorrhage. Eight hours later, shock. Eleven hours later, inverted uterus found. Condition poor for one month. Diarrhœa and pleurisy.	Vagina packed. Two transfusions given. Replacement four months later under general anæsthesia by combined vaginal and abdominal operation.	Uninterrupted recovery. Discharged well in fourteen days.
Aged 29. *5 para.*	*Post partum* hæmorrhage. Manual removal of adherent placenta. Inverted uterus found at time. No shock.	Immediate replacement.	Recovery.
Aged 27. *Primipara.*	Manual removal of adherent placenta. Diagnosed immediately. Shock.	Immediate replacement.	Recovery.
Aged 28. *3 para.*	Delivered 4.20 a.m. Baby 8½ lbs. Inversion found 4.40 a.m. Severe shock.	Intravenous injection of saline solution and glucose. Morphine. General anæsthesia and uterus replaced.	Died 5.50 a.m.

The treatment may be summarized as follows. The patient should be treated for shock and should receive intravenous injections of glucose and saline solution while a suitable blood donor is being found for transfusion. Morphine should be given, if necessary, for restlessness. The patient's head should be kept low. Bleeding should be controlled by packing; if the placenta is still attached, the packing should be done without separating it. Sepsis should be treated by means of gas-gangrene antiserum, vaginal douches and vaginal packing. It is wisest to wait till the patient has completely recovered from the shock and is convalescent before attempting replacement of the inverted uterus. I should wait for at least six weeks before attempting to replace the uterus, thus giving it a chance to undergo spontaneous reinversion. I have found on close observation of my patients that involution takes place quite normally in the inverted uterus, and at the end of six weeks the uterus is back almost to its normal size.

Operation.

The best operation is that of combined abdominal section, with pull on the inverted fundus with the volsellum and pressure from the vagina with the fingers or a sponge by an assistant. If this is not successful, a small incision in the posterior wall of the uterus at the level of the internal os releases the constricting ring and allows easy replacement. This operation leaves practically no weakness in the uterine wall, as opposed to the Spinelli operation, which splits the anterior wall of the cervix and the uterus, and has been followed by rupture of the uterus in subsequent pregnancies.

The operation should not be performed until the patient has quite recovered from her shock; there is then no more risk than there would be in any simple abdominal section. I would not operate on any patient earlier than six to eight weeks from the time of the inversion. The operation is no more difficult when it is delayed for a prolonged period, and there is always the chance of the inverted uterus spontaneously reinverting, this having occurred in three cases in this series.

Conclusions.

1. The treatment of the patient for shock is far more important than an attempt to replace the inverted uterus. Efforts at reinverting the uterus increase shock and waste valuable time.

2. Hæmorrhage and sepsis are not nearly such dangerous complications as shock.

3. The uterus may undergo spontaneous reinversion after involution has taken place, and should be given at least six to eight weeks to do so.

4. Failing spontaneous reinversion of the uterus, an operation for replacement can be done at any time later when the patient is fit and strong.

5. Delay for a long period (as much as four months in one of my cases) does not increase the difficulty of the operation.

6. The operation of choice is by the abdominal route, pressure being exerted from below; a small incision is made through the lower uterine segment and cervix, posteriorly if necessary, to release the constricting band. This operation is easy and devoid of shock, and it leaves very little subsequent weakness in the uterus for subsequent pregnancies.

Acknowledgements.

I am indebted to my colleagues at the Women's Hospital for access to the histories of their cases, and also to Professor Marshall Allan for help in preparing the slides.

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(2) G. H. Davis: "Acute Inversion of Uterus, with Report of Four Cases", *American Journal of Obstetrics and Gynæcology*, Volume XXVI, 1933, page 249.
(3) D. N. Barrows: "The Treatment of Recent Puerperal Inversion of the Uterus, with a Report of Five Cases", *American Journal of Obstetrics and Gynæcology*, Volume XXVII, 1934, page 105.

SOME EARLY REFERENCES TO TUBERCULOSIS IN AUSTRALIA.[1]

By J. B. CLELAND, M.D.,
Adelaide.

CAPTAIN COOK, on landing at Botany Bay in 1770, buried Forby Sutherland, a sailor, who had died of consumption. J. H. Maiden, in his "Biography of Sir Joseph Banks", suggests that this was probably the first white man buried in Australia. As far as eastern Australia is concerned this is probably correct, but it is possible that some of the early Dutchmen on the west coast of Australia died whilst their vessels were in that part of the world, and were buried on Australian soil. As far, however, as British interests were concerned in Australia, one of the first incidents was the burying of one of the crew who had been the victim of tuberculosis.

The exact nature of Forby Sutherland's illness, however, is not quite so simple as Maiden's statement might imply. Maiden apparently obtained his information from a paragraph in Arthur Kitson's "Captain James Cook, R.N., F.R.S., 'The Circumnavigator'", published in 1907, Maiden's work being published two years later. The paragraph (page 173) is as follows:

> Whilst here a seaman named Forbes ("Forby" in the muster roll) died of consumption, from which he had been suffering throughout the voyage, and was buried on shore on the point named by Cook in his memory, Point Sutherland. The anonymous pamphlet referred to above [apparently "an anonymous work on Australian discovery which was never completed", published in Sydney, and containing "a vague account of the landing, said to have been obtained from the blacks"] says that Cook does not give the cause of Sutherland's death, though he was usually very careful to do so, and then tells a story to the effect that Sutherland had found a metal plate affixed to a tree which showed that the Dutch had previously

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

been on the spot, and whilst securing it he had been fatally wounded by the blacks, and then Cook, in order to have the credit of being the first discoverer, carefully suppressed these facts, but that the plate was secured by some one or other, and is preserved in the British Museum. Unfortunately for the truth of this story Cook very distinctly states that Sutherland died of consumption, and, secondly, the plate is not in the care of the British Museum, nor can any trace of its existence be found in any records of the voyage.

The entry in "Captain Cook's Journal", edited by Captain W. J. L. Wharton, 1893, page 244, is as follows:

> Thursday, May 1st (1770): Last night Forby Sutherland, Seaman, departed this Life, and in the a.m. his body was buried ashore at the watering place, which occasioned my calling the south point after his name.

Here there is no reference to consumption. Captain Wharton states that Cook's "Journal" was in triplicate. His text was taken from Mr. Corner's copy "so far as it goes" (that is, until a few days before reaching Batavia—thereafter the Admiralty copy), paragraphs from the Admiralty copy, which do not appear in the former, being added, with a notation of their source. Kitson stated that:

> For the First Voyage I have consulted the "Log" of the *Endeavour* in the version so admirably edited by the late Admiral Sir J. W. L. Wharton (really W. J. L. Wharton).

Wharton, however, dealt with Cook's "Journal", and says that the ship's log book of the *Endeavour* is in the British Museum, and Cook's own log, not autographed, however, in the possession then of Mr. R. M. Hudson, of Sutherland.

There is no reference to Forby Sutherland's death in the "Journal of the Right Hon. Sir Joseph Banks", edited by Sir Joseph D. Hooker, 1896. There is no reference to consumption in Hawkesworth's account, and no light is thrown on the matter in the "Journal of Sydney Parkinson". Commander Francis J. Bayldon, in "Remarks on Criticisms of Explorers in the Pacific Ocean" (*Royal Australian Historical Society Journal and Proceedings*, Volume XIX, 1933, Part III, pages 158 to 171), deals exhaustively with those traducers of Cook who have hinted or stated that Cook was aware that he was not the first to discover the east coast of Australia, dismissing all such suggestions as without foundation. He does not refer to the anonymous pamphlet mentioned by Kitson.

It will be seen from these references that I had been unable to trace the source of Kitson's statement that Sutherland "died of consumption, from which he had been suffering throughout the voyage". The latter implies that there may be other references to his illness. It will be noticed that Kitson makes two inaccuracies. Captain Wharton published Cook's "Journal", not his "Log"; and Captain Wharton's initials are transferred.

At this stage the question was satisfactorily settled by a letter from Professor Ernest Scott, of Melbourne, to Mr. W. A. Cowan, the University Librarian, who had written at my request. He writes:

> There can be no doubt that Able-seaman Sutherland died of consumption. Cook says so plainly, in his Private Log. I copy out the words, with his spelling:
>
> > Tuesday, May 1 (1770) . . . Last night departed this Life Forby Sutherland, seaman, who died of a consumption (*sic*) and in the a.m. his body was entard (*sic*) ashore at the watering place. This circumstance occasioned my calling the south point of this bay Sutherland's Point.
>
> This is confirmed by Pickerell's Journal, under same date:
>
> > At 6 p.m. departed this life Forby Sutherland, seaman, of consumption, with which he had been afflicted ever since our departure from Streights le Maire.
>
> The statement that Sutherland died of wounds was printed in Bennett's "History of Discovery and Colonisation", the wounds, it was alleged, having been inflicted by natives. There is no contemporary authority for that allegation. If Sutherland had been wounded by natives Cook would surely have mentioned it, both in his official log and in his private Log.

Maiden mentions that Henry Kendall wrote a poem entitled "Sutherland's Grave" ("Poems of Henry Clarence Kendall", 1903, page 105). Underneath the title is "(The first white man buried in Australia)". This is a picturesque account of the surroundings of his grave and gives no account of Sutherland himself.

Afterwards, when settlement was established, persons from the Old Country whose lungs were believed to be "touched" came out to Australia, and whilst many of these recovered completely, or lived for many years, others died, and both categories were a potential danger to Australia. These persons were necessarily the producers of our indigenous cases of the disease.

Amongst tuberculous persons thus coming to Australia were those who came out more or less ill under definite medical advice; those who were suspected of having consumption, but who were still capable of living an active life, and came out more for precautional purposes than because the disease was well established; and, lastly, those who were compulsorily sent out from the Old Country—in other words, transported—and incidentally were suffering at the time from pulmonary tuberculosis. Thus Joseph Gerrald, one of the five "Scotch martyrs", transported in 1794 for what were really political activities, died in March, 1796, from consumption contracted in England, according to Collins (page 274). Three days later Skirving, also one of the "Scotch martyrs", died of dysentery.

Dr. George Bennett, speaking of Sydney in 1834 ("Wanderings in New South Wales", Volume I, page 340), says:

> A number of persons perish from that fatal disease consumption; but I do not regard it as produced by the climate, as it invariably attacks persons from England, of dissipated habits, or of employments uncongenial to health.

In 1835 we find the Quaker Backhouse stating, however, that consumption was not of frequent occurrence among immigrants, but children born in Australia of European parents sometimes died of it. ("A Narrative of a Visit to the Australian Colonies", page 229).

During the centenary celebrations in South Australia a very interesting historical play, by Max Afford, was staged, which centred around Colonel Light, the founder of Adelaide. His death from pulmonary tuberculosis, presumably from hæmoptysis, was almost too realistically portrayed. It seemed the general consensus of opinion that Colonel Light had died from pulmonary tuberculosis, and this led me to inquire as to the basis for such a belief. Fortunately, Miss Penelope Mayo is engaged in writing the life of Colonel Light, and she very kindly looked up notes for me and has epitomized these in the following account:

From April 1809-1814 Light saw service in the Peninsular War. In the course of this campaign he experienced much hardship and exposure to all weathers. His diary of the war has left on record that he was often without food for days at a time.

During the winter of 1824-1825 Light fell ill in Rome (possibly of malaria). In his diary of a voyage on the Continent, he refers in February 1825, to "the remnant of his long sickness" and to a violent cold in the head from which he was then suffering.

In his Log of the Steamship *Nile*, Light has left on record that in December 1834 he suffered from a suppurating throat and gout. The sailing of the *Rapid* for South Australia was delayed some weeks by his ill-health. A more rapid deterioration of his health followed upon arrival in South Australia, due in some measure to hard work, exposure to wet and cold, lack of suitable food, and mental stress. In April 1837, Light noticed and recorded in his Brief Journal, a change for the worse in his health. In an account of an expedition to Lynedoch in December 1837, Light recorded that he felt very unwell, was unable to sleep, and was suffering from gout.

By January 1839, when Light was engaged on a special survey near the Para River, he was utterly exhausted, troubled by asthma (?) and by a recurrence of feverish symptoms. In his last diary Light mentioned certain of his symptoms. In March 1839 he was suffering from dysentery (common in those days). On an expedition towards the Murray early in June 1839—a desperate venture for so sick a man—he had fever and ague, was unable to eat for some days, and returned to Adelaide in a high fever. In July he complained of a very violent cough and pains near his heart.

His colleague and friend, B. T. Finniss, mentions in his diary that at this time Light had a hectic flush and a cough. In letters written in 1838 and 1839, Light himself spoke of his shattered health, his rapid wasting, and his ten months' cough. His death, however, was quite peaceful.

Hodder, who had access to the Angas papers for material for his history of South Australia, states that Light died of consumption (Volume I, page 112).

Maria Gandy, who nursed Light for the last few years of his life, herself died of pulmonary tuberculosis in December 1847.

Although there is a reference to possible asthma, the account Miss Mayo furnishes leaves little doubt that Colonel Light actually died from pulmonary tuberculosis, which is almost confirmed by the fact that Maria Gandy, who nursed him, herself died from this disease a few years later. His death occurred on Monday, October 5, 1839.

Captain Charles Sturt, in his "Expedition into Central Australia", Volume II, page 238, published in 1849, makes this statement:

Whether it is owing to the properties I have described that the climate of this place (Adelaide) as also of Sydney, should be full of consumptive habits, I do not know, but in both places I have understood that such is the case and in both I have had reason to regret instances.

Dutton, in his "South Australia and its Mines" (page 102), refers to a report of the Colonial Surgeon, somewhere before 1846, that there were few cases of phthisis in South Australia.

J. C. Hawker, in "Early Experiences in South Australia" (1899, page 10), writing about October, 1838, of Mr. Bernard, Crown Solicitor, states: "The poor man was in consumption, and died about eighteen months after." Mrs. Harvey, wife of Dr. James Harvey, died apparently of "consumption" at Port Lincoln about 1843.

Mrs. Gouger, wife of Robert Gouger, died of consumption in Adelaide. On the other hand, some of our finest citizens, who lived to an advanced age, came out to South Australia for health reasons, in many cases on account of the suspicion of tuberculosis. Sir Samuel Davenport, for instance, was supposed, as a young man, to have had some symptoms suggestive of pulmonary tuberculosis; but as far as I know as a relative, he never manifested any symptoms in Australia, and lived in excellent health to the age of eighty-eight.

Hirsch, in his "Geographical and Historical Pathology", in Volume III, page 188, gives several references to pulmonary tuberculosis in Australia which may not be generally known. Thus there is an article in the *Transactions of the Epidemiological Society*, 1865, Volume II, page 85; an article by Richardson, *Edinburgh Medical Journal*, 1869, page 802; and a work by Thomson "On the Supposed Influence of Climate on Phthisis", Melbourne, 1871, 1879; and Reeves on "Consumption in Australia", Melbourne, 1874.

These short notes have a more particular application to South Australia, and seemed to me of sufficient interest to bring under the notice of members of the congress, in view of the fact that the question of tuberculosis in all its aspects is one of the major matters for consideration.

THE MANAGEMENT OF LARGE UMBILICAL HERNIÆ[1]

By L. M. McKILLOP, M.B., Ch.M. (Sydney), F.R.C.S. (Edinburgh), F.R.A.C.S., F.A.C.S.,

Honorary Surgeon to In-Patients, Mater Misericordiæ Public Hospital, Brisbane; Chairman, Treatment Committee, Queensland Cancer Trust.

WHILE a small and relatively minor hernia at or about the umbilicus may be an easy matter to repair and be followed usually by a perfectly satisfactory result, both immediate and ultimate, there are aspects of large herniæ in this situation which may tax the ingenuity of the most experienced of surgeons. One of these considerations is the length

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

of time that such a condition may have been allowed to exist, even in very large herniæ, before advice is sought. I have from hearsay known this to extend to a matter of forty-seven years. The sufferers from large hernia through, or immediately above, the umbilicus are usually middle-aged to elderly women of obese type and possibly suffering also from cardiac or bronchial symptoms. The primary cause is an inherent weakness in the closure and strengthening of the fasciæ in the region of the umbilical opening. In the ordinary course of life this may not betray itself, but, with the weakening of the abdominal muscles by the periodical stretching and thinning associated with pregnancies, followed later by an increase in the intraabdominal fat, progressive atony of the bowel (leading to overflatulence) and the development of chronic bronchitis or asthma causing cough and increased intraabdominal pressure, conditions come about which favour the more or less gradual protrusion of the sac and its contents through the weakened umbilical wall, usually just above the navel itself.

Finally, the hernia may reach such enormous dimensions that its sac may be found by some courageous surgeon to contain pounds of omental fat and feet of gut. Fortunately, the story is not always so harrowing as this, unless obstruction occurs even in a moderate sized hernia, when the surgeon may be faced with a formidable state of affairs.

Obviously, then, it will be advantageous to consider the management of herniæ at the umbilicus under three separate headings: moderate sized herniæ, very large herniæ, obstructed and/or strangulated herniæ.

Moderate Sized Herniæ.

If the circumstances of the case are such that the woman is likely to have further pregnancies or is unable to afford a sufficiently long post-operative convalescence or has asthma or a complication negativing surgical interference, a properly fitting support should be worn almost continuously, and steps should be taken to reduce the weight of the patient and the degree of intestinal flatulence by dieting and increased graduated exercises designed to improve the tone of the abdominal muscles. Should, however, the hernia be, or become painful, or should surgical interference be decided upon, what, then, is the best type of operation?

In any type of remedial operation three requirements must be fulfilled: (*a*) The sac only, but in its entirety, must be removed, after the return to the abdomen of any coils of gut on which no raw surface must be left uncovered. (*b*) The *fascia transversalis* should be carefully sewn and the recti muscles carefully approximated, that is, if they can be drawn together—a step not always possible—and the fascial edges overlapped from above or from side to side. (*c*) A firm support must be worn for at least two months after operation. My opinion is that in such a type of hernia where the opening is not very large, it does not greatly matter whether the sheath overlapping is done from side to side (Figure I) or by the Mayo method of sliding the

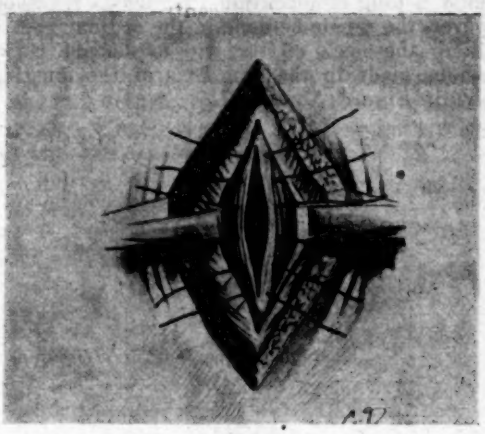

FIGURE I.

top leaf down in front of the bottom leaf and securing them to one another with fine kangaroo tendon, as shown in Figure II.

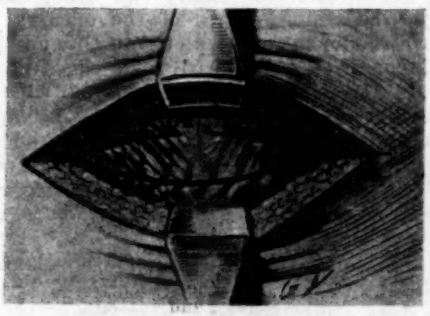

FIGURE II.

After the transverse elliptical incisions are made to encircle the navel (Figure III) the knife is carried down to and across the recti sheaths, and all

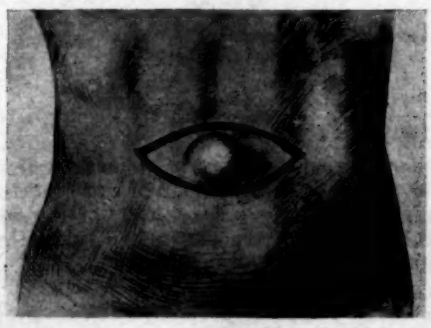

FIGURE III.

bleeding points are carefully controlled. The fascia to one side of the neck of the sac, which must always be carefully defined, is incised and the abdomen is

opened. The finger being used as a guide, the sac is then opened and a survey is made of its contents, usually a plug of omentum (Figure IV). Should, however, the repair be done in the ordinary vertical fashion, the neck of the sac is defined between incisions made in the long axis of the muscle, as shown in Figures V and VI.

FIGURE IV.

The omentum is then cut across and the proximal segment is very carefully ligated and dropped back (Figure VII).

The sac is then cut away, care being taken to keep to the defined edges of the opening. The abdomen should then be explored to note any pathological condition. The peritoneal edges are then sewn in eversion and the fascial edges are thoroughly overlapped by sutures of kangaroo tendon which bring one flap of sheath well across

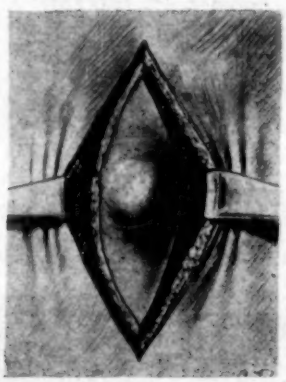

FIGURE V.

the underlying one. Silkworm gut mattress sutures are now placed so that the loop of each suture picks up the sheath of the rectus muscle of the opposite side and is tied on the skin after being threaded through a piece of rubber tubing.

Very Large Herniæ.

When the hernia is a very large one, conditions may be found which may be more difficult to manage. The patient's general condition may be unsatisfactory. For instance, she may be found to have an imperfect renal function or a fatty and dilated heart. Before what is often an unavoidable operation is undertaken, such medical defects must, as far as possible, be treated on general principles, and a prolonged rest in bed, with suitable dieting and the use of a firm abdominal support, may be called for. The sac is often multilocular, with processes which have insinuated themselves between the extraperitoneal fat and the rectus sheath, or between

FIGURE VI.

the latter and the skin; the omentum may be densely adherent therein and contained loops of bowel, usually transverse colon, may be adherent to the sac or to one another. In very occasional cases I have had to resect bowel to deal with the condition. The fascia may be so thinned out and the recti so

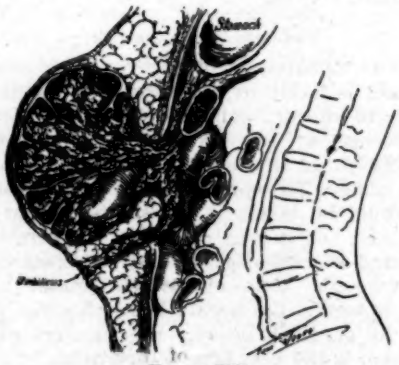

FIGURE VII.

far apart that it may be necessary to transplant a square of *fascia lata* to the deep surface of the thin aponeurosis, or to cut strips of *fascia lata* and employ them in the form of cross-lacing, as recommended by Gallie, of Toronto. I have been accustomed in such cases to employ several sutures of strong silkworm gut, passing them as mattress sutures threaded on fine rubber tubing from the skin and fascia of one side through the fascia and skin of the opposite side and *vice versa*. In this way the strain is taken obliquely by the recti and fascial sheath edges (Figure VIII).

In view of the size and vascularity of the wound, the outer edges should be drained for forty-eight hours with a rubber dam. It is a wise precaution to insert and leave a tube in the rectum and to order hypodermic injections of pitressin and eserine for two or three days after the operation. If these precautions are taken, morphine can safely be given if required, and the danger of ileus is probably lessened. In very large herniæ it may be justifiable, or even advisable, to reduce if possible the patient's weight beforehand. In our practice we have found the German preparation "Elityran" very useful, especially if combined with a reduction of the intake of fat and salty foods. The anæsthetic we employ nowadays is almost exclusively paraldehyde, given by the rectum, the use of which undoubtedly prevents post-operative shock and vomiting in practically every case.

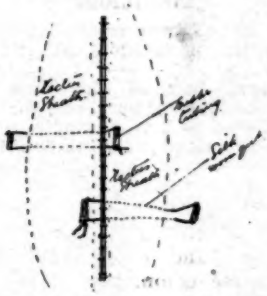

FIGURE VIII.

Obstructed or Strangulated Hernia.

When the hernia is of the obstructed or strangulated type the conditions are almost entirely different in that the first consideration is to save the life of the patient and to do one's best in the circumstances to repair the anatomical defect. Such a patient may be gravely ill and unable to stand much interference. After the abdomen is opened immediately lateral to the neck of the sac, its contents are identified and any adherent or constricted gut is examined for viability and defects in the sero-muscular coat, and such conditions are dealt with *secudum artem*. The peritoneum is very carefully closed after the great omentum has been drawn down behind the opening, if still available. If the patient's condition allows of it, an ordinary repair by overlapping or by fascial transplant is done; otherwise heavy silkworm gut cross sutures tied over rubber tubing are used as already described. In the event of the repair being done by the Mayo method, an extra precaution I have found useful is to bring the silkworm gut sutures obliquely up and down through the rectus sheaths, tying the knots through rubber tubing as shown in Figure IX.

Recently in *The British Medical Journal*, May 8, 1937, Wood Power has described a rather ingenious modification of the ordinary repair operation by incising each rectus sheath vertically at its outer margin for an extent greater than the hernial opening. He claims that this allows of the approximation of the recti muscle edges and of the overlying sheaths.

I have not yet tried this manœuvre, though I have on several occasions reflected the superficial sheath of one rectus and sutured it across the defect as one would turn the page of a book to one side. No instance has occurred of a hernia developing subsequently through the muscle so robbed of most of its sheath.

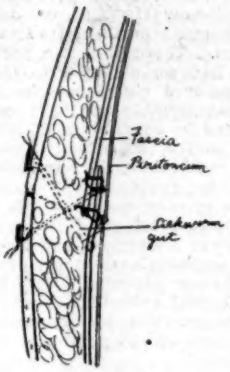

FIGURE IX.

After-Treatment.

As these patients are usually elderly, obese and very liable to the development of complications, it is essential that they be carefully nursed. After the return of consciousness the tightness of the binder should be adjusted for comfort and the patient turned from side to side and encouraged to take deep breathing exercises and to draw up the knees. All food likely to cause intestinal flatulence must be avoided, and the rectal tube passed regularly or left in. Pitressin and acetylcholine are useful for flatulent distension, and small doses of heroin or morphine for restlessness and pain. In the event of signs of ileus becoming apparent it may be advisable to give a spinal anæsthetic. Any complications which, from the nature of the case, might appear likely to arise, should be carefully watched for and, if possible, anticipated.

Results.

I cannot conveniently obtain with any accuracy my figures covering umbilical hernia operations since 1910, but for the 15-year period 1920 to 1934 I find that I have operated upon 33 umbilical herniæ. Of the patients, 6 died (embolism 1, intestinal obstruction 2, hypostatic pneumonia 2, diabetic coma 1). Of the remaining 27, 24 patients have remained well without any evidence of recurrence and 3 have had recurrence to a lesser or greater extent. The latter have exclusively been the cases in which obstruction was present and in which one could not be so deliberate in ensuring that the repair was perfectly sound.

Reports of Cases.

REPEATED EXTRADURAL HÆMORRHAGE.

By F. F. ELLIS, M.B., B.S.,
Resident Medical Officer to the Neurosurgical Department, Lewisham Hospital, Sydney.

THIS case is interesting in that it illustrates the occurrence of a severe intracranial calamity following a mild head injury.

The patient, a young man, aged sixteen years, was admitted to Lewisham Hospital on June 2 at 6 p.m. suffering from an acute cerebral trauma, the result of a motor-car accident. There were no positive neurological findings, but he had an abrasion to the right temporal region and a fractured right clavicle, and he was very pallid. He was comfortable and fully conscious until the next morning, when he complained of severe frontal headache. An hour and a half later he suddenly became unconscious. On examination he was found to be breathing stertorously and he could not be roused with painful stimulation. The plantar response was extensor on both sides, and there was a left-sided Oppenheim's reflex, and the upper and lower abdominal reflexes on the left side were absent. Hutchinson's pupil was present on the right side and there was a left-sided lower facial palsy. The diagnosis of right-sided extradural hæmorrhage was made, and immediate preparation for craniotomy was ordered. An X ray photograph of the skull was first taken, but no fracture was revealed.

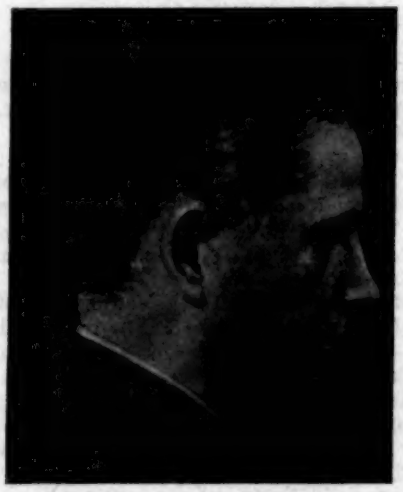

FIGURE I.

At operation a right subtemporal decompression was made, and a large well-formed clot about 3·75 centimetres (one and a half inches) in diameter was found extradurally overlying the face area of the motor cortex. Portion of the clot was sucked out, but no attempt was made to elevate the temporal lobe and to examine the floor of the middle cranial fossa. Two small meningeal vessels were stopped with endothermy, and the temporal muscle and scalp were closed over. An hour after operation the patient had roused sufficiently to answer simple questions, and he was quite well. Four hours later he became rapidly unconscious once again, with stertorous breathing, cyanosis and a very slow pulse. Immediate operation was again performed, the previous wound being opened. A clot was found extradurally between the temporal lobe and the floor of the middle cranial fossa. This was removed by suction, and blood was seen welling up from the medial aspect of the inferior surface of the temporal lobe. The decompression was widely opened, the brain was retracted, and a torn meningeal vein in the region of the *foramen spinosum* was located and stopped with endothermy. A muscle graft was inserted and the scalp was closed.

A concurrent blood transfusion was given with this operation, and the patient's post-operative condition was relatively good. A stormy convalescence followed, the patient being very restless for the next three days, but he showed no tendency to further bleeding. He was discharged three and a half weeks later with no evidence of any permanent ill effects from his extradural hæmorrhage.

The interesting features of this case are, first, the sudden unconsciousness appearing fourteen hours after relatively mild trauma, and second, the absence of fracture of the squamous temporal bone in the presence of severe extradural hæmorrhage.

Acknowledgements.

I wish to make grateful acknowledgements to Dr. Gilbert Phillips for permitting me to publish this case history, and also to Mr. Woodward Smith, who is responsible for the photograph.

Reviews.

TREATMENT OF HERNIA BY INJECTION.

THERE will always be those who desire at any cost to avoid an operation, and for such, and for those in whom operation is believed to be contraindicated, it is claimed that the injection treatment of hernia is suitable. Dr. Rice sets out in his book upon this subject his experience in the treatment of 476 cases in the hernia clinic at the Minneapolis General Hospital since 1932.[1]

An interesting historical survey of the subject in the first chapter, in which it is learned that within the last ten years several series of over 2,000 cases have been reported with a high percentage of successes, precedes chapters upon classification, anatomy, ætiology and diagnosis which in the main follow conventional lines and teaching. The ability, however, to palpate the impulse of the hernial sac, as apart from that of the hernial contents, or to palpate the fundus of the empty sac, is unfortunately denied to most practitioners. A most useful and well illustrated chapter is given to the description of the various forms of truss and their application.

It may be mentioned here that although the treatment is ambulatory it is essential that a truss be worn for a short time before injections are started, day and night during the series of injections and for a minimal period of three months thereafter.

A description of the technique employed and of the solutions favoured is followed by a discussion of the general indications and contraindications for this method. Whilst exception may be taken to the statement that operation in early childhood and infancy is very liable to be followed by recurrence, there will be complete agreement with the warning, repeated on several occasions, that inability to reduce a hernia completely and to maintain reduction constitutes an absolute contraindication to use of the method. Other factors rendering success unlikely are, a very large external ring, obesity, chronic cough or constipation, or prostatic hypertrophy.

The general principle involved is that by the injection of sclerosing fluids it is possible to produce so much scar tissue formation in and around the inguinal canal or other site of hernia that the sac is more or less obliterated and the region of the hernia itself so thickened and strengthened that abnormal protrusion is no longer possible. The author believes, nevertheless, that removal of the sac alone by surgical approach is ineffective.

It is evident that the author feels himself on more secure ground when dealing with oblique inguinal hernia than with any other variety, although full details are given of the treatment of such, even of post-operative ventral hernia.

[1] "Injection Treatment of Hernia", by C. O. Rice, M.D., F.A.C.S., with the assistance and cooperation of H. Mattson, M.D.; 1937. Philadelphia: F. A. Davis Company. Demy 8vo, pp. 275, with 83 illustrations. Price: $4.50 net.

In a frank discussion it is learned that complications and sequelæ are surprisingly few; abscess formation, not uncommon in other reported series, occurring in only two instances. The series of operation cases selected for purposes of comparison conveys the impression that the choice of surgical clinic was somewhat unfortunate.

The histopathology of injection treatment is considered at length and a process of fibrosis is demonstrated, but no discussion is found as to the usual fate of fibrous tissue when subjected to continuous strain.

The chapter which most intimately concerns the practitioner is that on end-results. A number of series have previously been reported with from 94% to 100% of successes, and Dr. Rice considers that after eliminating 97 patients who did not receive sufficient treatment or who did not return for a final survey, cure was obtained in 97·6% of cases in his clinic. It is not stated how many of these cures were brought about in hernias other than the oblique inguinal type.

The final chapter deals rather surprisingly with the medico-legal aspects of hernia as viewed in the various States of the Union, and is followed by quite a comprehensive bibliography and an index.

OPERATIVE MIDWIFERY.

"OPERATIVE MIDWIFERY" by Munro Kerr appears in its fourth edition under the new title "Operative Obstetrics", and has been almost entirely re-written by this distinguished Glasgow obstetrician with the help of two former assistants, Donald McIntyre and Fyfe Anderson.[1] It can be stated at once that here the obstetrician will find guidance in detecting abnormalities, in correcting them, and in treating emergencies. The form and arrangment follow those of previous editions and the subject matter is treated in the bed-side manner rather than under comprehensive classification. This approach, together with the author's personal experience and advice, makes the book essentially practical and easy to read.

The obstetrical outlook has completely changed in the last forty years, although no difficulties occur today which were not known to Herman. Much of this is due to antenatal supervision which Munro Kerr considers "the greatest advance which has ever occurred in the whole history of obstetrics". The great importance of a thorough examination of all patients at the thirty-fourth to thirty-sixth week is stressed, and the author approves of routine X ray examination when doubt exists concerning the presentation and disposition of the legs.

A protest is raised against the increasing tendency to rupture the membranes in uterine inertia. Postural treatment is preferred to Buist's pads for correcting an occipito-posterior position.

Conservative measures should not be pushed beyond rational limits, and the judicious use of forceps at the outlet and the performance of a timely episiotomy are advocated. The Burns method of breech delivery is mentioned favourably, and the forceps are used on the after-coming head if the least difficulty is experienced.

Disproportion, and particularly the merits of "a trial of labour", are treated in a masterly fashion. The author favours trial labour in all *primigravidæ* with mild to medium disproportion and condemns induction. This subject has been much discussed in recent years, and Munro Kerr is in accord with American, Continental and most British authorities. Cæsarean section with its growing list of "relative" indications is discussed in detail. Its promiscuous employment by the "obstetrically ill-educated and inexperienced" is condemned. The suture material favoured is catgut with a thin silk thread incorporated on its surface (McIntyre): Chromicized gut is used in the lower segment operation.

The importance of avoiding intrauterine manipulations in delivering the placenta is stressed. The old adage "once a Cæsarean section, always a Cæsarean section" does not apply in every instance if the lower segment operation is performed. Spinal anæsthesia is the ideal anæsthetic for the lower segment operation. Abdominal hysterotomy for rapid evacuation of the uterus is advised after the twenty-fifth week, but many authorities today prefer this route from the sixteenth week.

The indications for craniotomy are now much curtailed, but let it be remembered: "It is quite incorrect to assert that craniotomy is about as dangerous to the mother as Cæsarean section." Manual removal of the placenta is a dangerous operation, and the injection of sterile saline solution through the umbilical vein should be tried first.

Mention is made of the uses of "Amniography" for radiographically demonstrating the position of the placenta in suspected cases of *placenta prævia*.

The time-honoured treatment of packing the vagina for bleeding due to *placenta prævia* or accidental hæmorrhage is not favoured. Recent figures from all clinics testify to the great reduction of maternal and fœtal mortality in *placenta prævia* treated by Cæsarean section. Accidental hæmorrhage, on the other hand, is best treated conservatively, the essentials being rest, warmth, morphine and blood transfusion.

The work of Holland on intracranial injuries of the newborn is stated, and the treatment of *asphyxia pallida* by intratracheal inflation and oxygen administration (Blaikley and Gibberd) is mentioned.

Throughout the book all the varied treatments advocated and employed are given, and new methods stand or fall by the final test of effect on maternal and fœtal mortality and morbidity. The inspiration of men whose names have been great in obstetric history is apparent, but it can also be said that modern workers receive due credit and justice.

[1] "Operative Obstetrics: A Guide to the Difficulties and Complications of Obstetric Practice", by J. M. Kerr, LL.D., M.D., F.C.O.G., with the assistance of D. McIntyre, M.D., F.C.O.G., and D. F. Anderson, M.D.; Fourth Edition; 1937. London: Baillière, Tindall and Cox. Royal 8vo, pp. 860, with 338 illustrations. Price: 45s. net.

DERMATOLOGY.

THE fourth edition of Robert W. MacKenna's "Diseases of the Skin" has been revised and enlarged by his son Robert M. B. MacKenna as was the last edition.[1] A very complete revision in this edition has resulted in a general all-round improvement.

We are glad to note that metric quantities or percentages are given in the prescriptions as alternative to the British units.

The practitioner will find an excellent detailed treatment for impetigo and will note that "Elastoplast" is advocated for selected cases.

That sulphonamide is suggested as of use in the treatment of erysipelas is indicative of the extent of the revision. The illustrations and the text covering the diagnosis are particularly good.

It is rightly pointed out that there should be no routine treatment for syphilis; but the scheme of treatment given and devised by the late R. W. MacKenna does not fulfil modern requirements in that intermission in the earlier stages of treatment is advocated. In view of the recent international reviews of the treatment of extremely large numbers of cases, it is abundantly clear that there must be no intermission in treatment in the first year.

Sound lines of treatment are suggested for tinea of the foot, which will be of special use and interest to the Australian practitioner.

The discussion on the ætiology of the common seborrhœic dermatitis is probably the best short description that has yet been published in the English language.

A very interesting and unorthodox account is given of the life history of *Sarcoptes scabei*; this is noted as being derived from the work of Professor W. S. Paton. A strong warning is sounded as to excessive use of sulphur in scabies, which could with advantage be read by all who have occasion to treat scabies.

Allergy is clearly and briefly described. Bismuth is still preferred by the author to gold salts in the treatment of

[1] "Diseases of the Skin: A Manual for Students and Practitioners", by the late R. W. Mackenna, M.A., M.D., Ch.B.; Fourth Edition, revised and enlarged by R. M. B. MacKenna, M.A., M.D., M.R.C.P.; 1937. London: Baillière, Tindall and Cox. Royal 8vo, pp. 572, with 168 illustrations and 46 coloured plates. Price: 20s. net.

lupus erythematosus. The Cranston Low classification of dermatitis into sensitization and non-sensitization dermatitis is broadly followed. This classification avoids the word eczema with all the attendant difficulties of definition.

In the present edition rodent ulcer is adequately described, but, again, free excision, if cosmetically possible, is given as first therapeutic choice. Surely in these times some form of radiotherapy should be chosen whenever possible. The only treatment suggested for squamous epithelioma is excision.

Useful chapters on diseases of the hair and nails are included. An excellent selection of coloured plates and photographs appear throughout. These illustrations are superior to those appearing in any current British dermatological text-book. The index is full and a chance examination of fifty entries proved correct.

MICROSCOPIC TECHNIQUE.

WORKERS in animal and plant tissues will welcome the second edition, revised and enlarged, of the "Handbook of Microscopical Technique" by McClung.[1] The various chapters have been written by selected contributors, thirty-four in all, many of whom are professors of leading American universities. Representatives of the Universities of Oxford, Montreal and Stockholm are also included. In the new edition an effort has been made to bring up to date the methods described and to give accounts of new methods and types of apparatus. The dioxan technique for paraffin sections, directions for free hand manipulation of living material, methods of staining *boutons terminaux*, the fused quartz rod method of illuminating living structures, microincineration and fluorescent microscopy are dealt with in considerable detail and many useful practical hints are given, which facilitate the application of these modern methods. There is also a description of the centrifuge microscope.

The number of pages has been increased from 495 to 698, a sufficient indication in itself of the growth of the subject. There are 82 illustrations which have been very carefully chosen. Diagrams such as those on pages 667 to 669 relating to the centrifuge microscope are extremely helpful and make further description unnecessary.

The chapter on fluorescent microscopy is rather short and will need to be amplified in a future edition. A more precise definition of fluorescence and a fuller account of the phenomenon will be appreciated by those who have not had the opportunity of studying the deeper aspects of the subject in the text books of physics. Photographs showing the power of the method, such as are given in Danckworth's "*Lumineszenzanalyse*", would stimulate interest in this type of work, which is still far too little used.

In the chapter on the microincineration method of demonstrating mineral elements in tissues the use of the photoelectric cell in a dark-field photometer apparatus is carefully described. Here again it would have been of help to the research worker to have had the details (or diagram) of the Du Bridge and Brown amplifier placed at his disposal instead of being referred to the original paper in the *Review of Scientific Instruments*.

High praise must be accorded to the authors of the chapters dealing with histological methods of studying red blood cells and leucocytes. Every aspect of this subject is dealt with. On page 312 there is a useful chart for calculating the rate of sedimentation of red blood cells for varying hæmatocrit values in terms of a hæmatocrit value of 45. A method is also given for determining the blood group when only one blood of Group II (Jansky) or of Group III (Jansky) is available. Nine different solutions are given for diluting blood platelets.

Enough has been said to indicate that this encyclopædic work is invaluable to anyone who wishes to acquire a thorough microscopic technique. No part of the subject has been overlooked. The volume is excellently printed and produced. The illustrations approach photographic perfection, for example on pages 487 and 488, where *boutons terminaux* are depicted. No student of microscopy can afford to be without this book.

[1] "Handbook of Microscopical Technique for Workers in Animal and Plant Tissues", edited by C. E. McClung, Ph.D.; Second Edition, revised and enlarged; 1937. New York: Paul B. Hoeber Incorporated; Australia: Angus and Robertson Limited. Medium 8vo, pp. 715, with 82 illustrations. Price: 45s. net.

MINOR AILMENTS.

"MINOR MALADIES", the seventh edition of which is under review, may be listed among the classics of medical literature.[1] In its thirty years of being its content has altered, new matter has replaced older matter, the viewpoint of the author has changed to some extent and his lifetime of experience has given fresh argument to the original thesis. In essentials, however, it remains a text-book descriptive of those lesser ailments which medical practitioners are often called upon to treat, but to cope with which they are often ill-equipped.

The earlier editions are characterized by an egotism, a forceful presentation of the author's somewhat heterodox beliefs, and, not least of all, by occasional facetious alliteration, as, for example, when he is considering constipation, discussing defæcation or evaluating evacuation. There is stimulus to thought in many of his views and an abundance of aphorism for personal experience to verify or disprove. The following are illustrative: in his own experience the assumption of the ventral decubitus will always cure cold feet; removal of butchers' meat from the dietary will ultimately cause the disappearance of pustules in acne; a patch worn over one eye will often prevent sea-sickness. Some aphorisms he takes from other writers: "oversexed men show a stronger tendency to baldness than the normally sexed": "a pustular eruption on the back only and practically confined to the skin overlying the trapezius on either side is caused almost certainly by syphilis or tubercle": "emetine hydrochloride injected intramuscularly will often cure boils": "for the erythema of sunburn apply a saturated solution of epsom salts leaving it to dry on": "four soda mint tablets or a dose of epsom salts will relieve paroxysmal sneezing": "diffuse greying of the hair in relatively young people is usually attributable to eyestrain".

The principal alteration in the present edition is the inclusion of a chapter on "Minor Dietetics". To make room for it here there has been some re-arrangement and exclusion of other parts of the text. On the whole this new chapter is rather disappointing. Much of it has already appeared in earlier editions. The indictment of the polysaccharides is repeated and the author's condemnation of the casserole and cooking pot is renewed. The cooking pot may, however, continue to bubble on the hobs of the fastidious who have heard rumours of the methods of "intensive cultivation" employed by Chinese market gardeners. Before yet "the grinders cease because they are few", and when the gastro-intestinal mucosa is docile, the advice to eat turnips, carrots, artichokes and other tubers in the raw state is no doubt good, but some of us have suffered both agony and shame after herbivorous adventures. It is becoming recognized, perhaps tardily, that the amount of disaccharide consumed as sweets of various kinds, especially by children, is for many reasons deplorable, and that the benefit to be derived from fruit and vegetables, which are the natural, pleasant and sufficient sources of body sugar and other vital items of diet, is largely nullified by prolonged cooking. But when we observe the modicum of fruit and/or vegetable necessary for the maintenance of apparently perfect health, we become impatient with such wild statements as that the cooking of food and the sterilization of milk by heat "filled the wards and out-patients rooms of the hospitals, breeding criminals, pansies and village idiots".

It is a pity that a book in so many respects invaluable should be spoiled by wild statements in support of views that can be regarded only as fanatic.

[1] "Minor Maladies and their Treatment", by L. Williams, M.D.; Seventh Edition; 1937. London: Baillière, Tindall and Cox; Australia: Angus and Robertson Limited. Crown 8vo, pp. 451. Price: 16s. net.

The Medical Journal of Australia

SATURDAY, FEBRUARY 5, 1938.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE CONTROL OF NARCOTIC DRUGS IN NEW SOUTH WALES.

DRUG addicts are made more by accident than by inherent viciousness, though many hold that one certain type of individual is more prone to addiction than another. If addiction to drugs is to be prevented, and most people will agree that an effort must be made to protect people against their own foolishness or vice, legal enactments have to be enforced so that drugs likely to cause addiction can be supplied only under certain specified conditions. Fortunately, drug addiction is not a serious menace in Australia; but deaths by misadventure (as by an unintentional overdose) or deliberate suicide are still far too common.

In the *Police Offences Amendment (Drugs) Act*, 1927 (generally known as the *Dangerous Drugs Act*), the New South Wales authorities made a praiseworthy effort to check narcotic drug addiction as well as fatalities from accidental overdosage of certain drugs and deliberate self-destruction. The drugs of which the original Act took cognizance were morphine, diamorphine (heroin), cocaine and ecgonine. Additions to the provisions of the Act have been *Cannabis indica*, paraldehyde and all barbiturates.

In some particulars the wording of the Act is deficient. Any ambiguity or uncertainty in the Act or regulations should be rectified as promptly as political exigencies permit. For instance, regulations under the Act may be actually *ultra vires* the Act. Dionin does not occur naturally in opium. It is an artificial alkaloid (ethyl morphine). In the event of addiction following the use of dionin it is doubtful whether this drug could be held to be provided for by the Act. The position of codeine is even worse. Codeine occurs naturally in opium; but it is, in composition, methyl morphine and can also be made from morphine. Fortunately, codeine addiction is not sufficiently common to be a serious social evil, and apparently it is not considered to be controlled by the *Dangerous Drugs Act*. Another pertinent question is whether barbiturates are genuinely addictable in the same way as is morphine or cocaine. In the event of a prosecution, a court might hold that they were not. We know that barbiturates may be taken regularly and in increasing doses; but that is not convincing proof that they are genuinely addictable. By proclamation the Governor may add to the schedule of the Act any drug of whatever kind which is, or is likely to be, productive of ill-effects substantially of the same character or nature as or analogous to those produced by morphine or cocaine. The question is whether the repeatedly sought hypnotic effect of the barbiturates can be held to be comparable to the addiction caused by the use of cocaine, morphine or dionin. Eminent legal authorities have maintained that the two groups are not comparable, and that a prosecution concerning any of the barbiturates would not be successful.

Three or four aspects of the problem require serious consideration. The first is that at one time the Pharmacy Board administered the Act and regulations; but subsequently the Police authorities obtained control. Whether this change was for the better is not by any means clear; we do not think it was. In particular, when we consider the immense

series of barbiturates, it is doubtful whether the Police authorities would be able to identify by name even a substantial proportion of them. The fact that many preparations are barbiturates concealed under other names may be entirely overlooked. A recent advertisement extolling the virtues of a preparation, expressly denied that it was a barbiturate, and stated that it was a compound of another substance, which is merely a synonym for barbituric acid.

In the second place, private hospital practice presents a vexing problem. Doctors have been known to order amounts of morphine, heroin and barbiturates in excess of those actually used, the unexpended balance being left at the hospital. It does not take long for quantities to accumulate and sinister possibilities arise which may result in addiction, death by misadventure or deliberate suicide. It would appear that the barbiturates are controlled only by the *Dangerous Drugs Act*. The *Poisons Act* does not apply to barbiturates. It should be incumbent on the proprietors of private hospitals to have drug licences, and there should be rigid supervision, particularly in reference to barbiturates.

The third point that needs emphasis is the obligation on medical practitioners to conform to the strict letter of the law. It is obviously unfair to ask any pharmacist to dispense drugs coming within the category of dangerous drugs by a telephonic communication on the pretence that the circumstances of the case are urgent; nor should telegrams be used for this purpose. Such a procedure is tantamount to asking the pharmacist to break the law and render himself liable to a penalty. Prescriptions should always be written in conformity with the Act. It has often been objected that the use of "Luminal" for epilepsy over long periods of time would be greatly hampered by strict observance of the provisions of the Act. Such an objection is purely fanciful, and it cannot be too strongly insisted upon that the Act imposes no actual hardship on members of the medical profession or on legitimate patients. Grievances are more fancied than real. The provisions of the Act and regulations should be faithfully observed. Indian hemp is not a serious menace in Australia. Addiction to paraldehyde is uncommon; but it does occur, in spite of the obnoxious, nauseous taste of the drug. As in other cases of addiction, those who take it will steadfastly deny their consumption of the drug, even if their persons and habitations reek with the odour of it.

A fourth matter that should be dealt with in a stringent fashion is the free sample custom. Medical practitioners are assailed with free samples of the various barbiturates. In the course of a year these assume formidable proportions. Practitioners are also invited to apply for similar free samples of some of the newer morphine and codeine derivatives. There would appear to be no check whatever on this practice. The samples may reach the doctor or be intercepted by someone in his or other employ. There would be no means of tracing the samples. It should be made an offence to post or otherwise to deliver such samples, except on a definite order, and the receipt of such samples should be specifically acknowledged.

Finally, any loopholes, inaccuracies or inconsistencies in the Act should receive the immediate attention of the authorities.

Current Comment.

MEDICINAL TREATMENT OF ANGINA PECTORIS.

For some years the general opinion of physicians has been that vasodilator drugs are the most efficacious in reducing the number and severity of anginal attacks. More recently, Joseph E. F. Riseman and Morton G. Brown[1] have attempted an accurate evaluation of the efficacy of therapy with a number of different agents. In this investigation the authors have studied the results of treatment while the patients performed standardized exercise tolerance tests. All the patients, of whom there were twenty-six, were suffering from no disease other than that of the coronary arteries, and in all cases electrocardiographic tracings showed changes consistent with coronary disease. Twenty-one of the patients were males and five females. Approximately half of the subjects were able to perform light work, although all had experienced attacks of *angina pectoris* on exertion.

[1] *Archives of Internal Medicine*, July, 1937.

All the patients were observed in a specially constituted clinic, and fifteen drugs were employed. In the use of digitalis, the appropriate dose in relation to body weight was calculated according to the method of Eggleston; but the doses of all the other drugs were the same for all patients, each patient receiving one particular preparation for a full week before its effects were evaluated. Once there was reasonable evidence that a given drug was exerting a favourable influence, it was discontinued and an inert tablet of the same appearance was substituted for it.

The benefits of the various methods of medication were assessed by attention to the patients' subjective sensations and by keeping records of the actual number of attacks experienced during the course.

It may appear strange to record that on clinical evidence alone, and after inquiry into the subjective sensations of this group of patients, lactose and sodium bicarbonate produced beneficial results just as frequently as other drugs, though the total number of anginal attacks during a given period was not reduced. Nevertheless, an improvement in the patients' outlook, a sense of added well-being, a freedom from apprehension and anxiety (doubtless increased by the knowledge that the subjects were under constant medical supervision) were undoubtedly apparent after the exhibition of these inert substances. All this confirms the findings of certain English investigators in 1935: "A measure of improvement appears to result from every remedy tried, and at least as great an improvement during treatment with *placebo*." But no patient was able to do more work after taking lactose or sodium bicarbonate, and any real improvement in exercise tolerance—the most valuable check upon the efficacy of therapy—at once disappeared when either of these two drugs was taken. Of the twenty-six patients, nine remained unrelieved in any way by any of the fifteen drugs used.

Riseman and Brown found that in the absence of all medication, the work which their patients were capable of performing before pain was evident was extraordinarily constant under the standardized conditions of exercise used in their studies, and they concluded that any change for the better which regularly appeared after a certain drug was given, and which disappeared after its omission, must have been produced by the use of that drug.

The conclusions of Riseman and Brown may be summed up by saying that the patients' own estimation of the therapeutic good wrought by medicinal treatment indicated that all drugs were approximately equal in value, and that they derived just as much benefit from the veriest *placebo* as from other medicaments. But standard exercise tests proved that those patients treated with such agents as tissue extracts, potassium iodide, lactose or sodium bicarbonate, were unable to perform any more work than was possible without any medication. The exhibition of glyceryl trinitrate (trinitroglycerine), however, given before exercise began, permitted a considerable increase in the amount of the patients' effort. This prophylactic effect was often of short duration, but patients could often be freed from attacks in daily life by taking glyceryl trinitrate in hourly doses; and small doses (one five-hundredth of a grain) were found to be as valuable as larger amounts.

Some 50% of the patients felt an improvement after taking aminophylline, quinidine sulphate, theophylline, calcium salicylate, erythrol tetranitrate and atropine sulphate, but were rarely capable of increased effort on using barbiturates or preparations of codeia. Sodium nitrite and dinitrophenol were very rarely found to be effective. The same was found with digitalis; this drug, indeed, was often found to produce a striking increase in the number of anginal attacks.

In general, the findings of Riseman and Brown bear out those of Hoyle and Evans, published in 1934. The latter authors then stated that glyceryl trinitrate was the most efficacious drug in reducing the number and severity of attacks of *angina pectoris*. They advised doses of one one-hundredth to one twenty-fifth of a grain in tablet form, the tablet to be slowly chewed and not swallowed whole. By this plan, they said, 86% of patients were relieved. The drug produced no harmful effects in any subject, and was used freely in some cases for upwards of two or three years, some sufferers being able to undergo more physical exertion and to lead a fuller life than had been possible before.

ORBITAL CELLULITIS DUE TO SINUS INFECTION.

ALL practitioners to whom the journal is available would do well to read the account of a discussion on orbital cellulitis due to sinus infection, published in the September, 1937, issue of *Proceedings of the Royal Society of Medicine*. E. D. D. Davis, the opener, discussed fifty-four cases. Of these, twenty-four had followed frontal sinus and fifteen ethmoidal sinus suppuration. He drew attention to the danger of the condition, but at the same time showed that there was a considerable variation in its severity. When an orbital abscess is present simple external operation should not be delayed; drainage should be established with counter-drainage in the nose. When œdema of the orbit is great, the "more serious" condition of cavernous sinus thrombosis is to be feared. The treatment of an orbital abscess arising from the nose is always urgent, and in Davis's opinion more urgent than acute inflammation of the mastoid process. Thirty-four of Davis's thirty-seven patients treated by the external operation recovered.

S. H. Mygind, who took part in the discussion, recognized three stages of the condition: simple swelling of the orbit, periorbital abscess and inflammation of the intraorbital tissue proper. He emphasized the need for thorough opening up of tissues, and described the condition as most dangerous when the tissues of the orbit itself were involved.

Abstracts from Current Medical Literature.

MEDICINE.

Primary Friedländer Pneumonia.

S. SOLOMON (*The Journal of the American Medical Association*, March 20, 1937) has reported 32 cases of pneumonia due to Friedländer's pneumobacillus. The pneumonia occurred mainly between the third and seventh decades. Males were affected in the proportion of seven to one. Cold, exposure, alcoholism and malnutrition were predisposing factors. The onset was abrupt in 75% of cases. Chill, pleuritic pain, cough and malaise were usual. Dyspnœa and cyanosis occurred. The temperature ranged between 37·8° and 41·1° C. (100° and 106° F.), but was often not above 38·8° C. (102° F.). The course was fulminating; death occurred from the second to the sixteenth day, usually preceded by pulmonary œdema. Of the patients in this series 97% died. The consolidation was lobar in 27 cases and lobular in four. In only half the lobar cases were the signs those of frank consolidation, dulness, faint breath and voice sounds and a few râles being the only signs. The sputum was characteristic—a thick mixture of blood and mucus, brick-red and homogeneous, like chocolate pudding, though of a redder hue. A direct smear revealed the organism. A smear on agar plate incubated for twenty-four to forty-eight hours showed slimy grey colonies. Lung puncture with the aspiration and culture of lung juice revealed the organism in ten cases. Blood cultures were positive in 73% of cases, all ending fatally. The leucocyte count revealed absolute or relative leucopenia in 63%, less than 6,000 white cells per cubic millimetre in six cases, and between 6,000 and 12,000 in nine others. Complications were similar to those of pneumococcal pneumonia, gross sloughing of the lung, abscess and meningitis being most frequent. Treatment was as for other forms of pneumonia. Specific horse serum for Type A Friedländer's pneumococcus was used in five cases; all ended fatally. The author maintains that these were all primary cases of Friedländer's pneumonia.

Ménière's Disease.

W. E. DANDY (*The Journal of the American Medical Association*, March 20, 1937) discusses the pathology of Ménière's disease. He has previously reported that this condition can be cured by section of the auditory nerve or of the vestibular branch alone. The cause was found in some cases to be gross intracranial disease. The symptoms were deafness, tinnitus and attacks of severe vertigo. Seven cases are recorded; in two of these tumours were found in the cerebellopontine angle, and in one an aneurysm of the basillar artery. In five cases large arterial loops from the anteroinferior cerebellar artery were found in the lateral cistern; these loops acted by strangling or compressing the auditory nerve. These loops are congenital and it is suggested that they produce effects mainly in middle age or later, because the arteries become more rigid with age. It is probable that in many cases smaller arteries cause compression of the nerve in a similar way—possibly some 30 or 40 cases in the author's series of 160. In bilateral Ménière's disease there must be some intrinsic lesion of the auditory vestibular pathways in the brain stem, and this doubtless accounts for some of the unilateral disorders. It is known that a nerve, once damaged, still remains a potential source of subsequent attacks of Ménière's disease.

The Contagiousness of Lobar Pneumonia.

J. E. BENJAMIN *et alii* (*Annals of Internal Medicine*, September, 1937) state that they are convinced that lobar pneumonia is a contagious disease. In the course of their experiments in the typing of pneumococci they have observed not a few instances of cross-infection with pneumonia in their wards, and of pneumonia of identical type occurring in their patients' visitors. They have records of two and more members of families contracting pneumonia due to the same type of pneumococcus. They have successfully instituted precautions to prevent the spread of pneumonia. Patients with pneumonia are segregated in cubicles, and physicians and nurses are strictly enjoined to wear gowns and masks and to wash their hands after each examination and treatment. The authors recommend that pneumonia should be made a compulsorily notifiable disease.

Silicosis.

J. J. DENNY, W. D. ROBSON AND D. A. IRWIN (*Canadian Medical Association Journal*, July, 1937) discuss the prevention of silicosis by metallic aluminium. It was found that guinea-pigs exposed to dust containing 35% of free silica for up to a year did not develop silicosis, though at autopsy much dust was found in the lungs. It has been shown that the fibrosis of silicosis is due to the chemical reaction and not to the physical presence of silicious material. Particles of quartz of one to three microns produce an acute proliferative fibrotic response, while particles of ten to twelve microns cause only a foreign body response. It is found that the presence of small amounts of metallic aluminium prevent silica from passing into solution. Rabbits were dusted with silica, and a control group with silica *plus* less than 1% of metallic aluminium. The control group developed silicosis, while the latter group did not. Further experiments showed that silica from different sources varied greatly in solubility, and that the solubility of quartz was reduced by large amounts of calcium hydroxide, but was entirely dependent on the strongly alkaline reaction. In the rabbits pathological investigation showed that there was more quartz in the lungs of the aluminium-treated animals than in the control group, but, owing to the action of the aluminium, the latter showed degeneration, giant cell formation and migration of the dust cells into the pulmonary lymphatics, with elongation of dust cells to become fixed tissue cells. These changes are typical of silicosis. In those rabbits treated with aluminium the dust cells showed little degeneration, much less tendency to giant-cell formation and lymphatic migration, and practically no elongation. This is the type of reaction seen when relatively innocuous dusts are present in the lung.

Rheumatic Heart Disease in School Children.

JACOB M. CAHAN (*Annals of Internal Medicine*, June, 1937) has reviewed the histories and examinations of 391 cases of rheumatic heart disease in school children, of whom 214 were boys and 177 were girls. In 79 instances there was a family history of the condition. Tonsillitis occurred in 40% of cases, and from the data available on this point the author infers that the popular operation of tonsillectomy is often inadequate to prevent or to ameliorate all cases of chorea, acute rheumatic fever or rheumatic heart disease. Acute rheumatic fever was recorded in 18·6% of the histories, the first attack occurring most often at the age of seven years; the number of attacks varied from one to five. Chorea occurred in 11%, the first attack occurring most commonly at eight years of age. Other symptoms noted in the anamnesis were as follows: joint pains, swelling of joints, recurrent unexplained abdominal pains, enuresis, severe muscle or growing pains and epistaxis. There were 191 cases of organic heart disease; 113 patients showed abnormal signs or symptoms referable to the heart, but the diagnosis of heart disease was uncertain (Class E). Forty-six children were classed as having no circulatory disease and it was thought advisable that these should be examined periodically because of the presence or history of an ætiological factor which might cause heart disease (Class F). In the first group there were 118 diagnoses of valvular defects, 44 of hypertrophy of the heart with chronic adhesive pericarditis; in the remainder congenital defects were diagnosed. There were four cases in which the conduction systems were pathological. The problem of correlating the functional capacity of the child suffering from heart disease with the restriction of physical activities in the school is

freely discussed. A classification of the restrictions is given as a useful guide to determine the activities of any individual child. The question of the activity of a rheumatic heart condition is of the first importance in deciding whether the child is allowed to attend school or return to school after an acute illness; various criteria for this determination are listed. In general it is considered that the condition of many children with cardiac lesions becomes aggravated during the average twelve years of education; on the other hand, there are many pupils who go through their school life without any aggravation of their cardiopathy. The prognosis of the child with heart disease depends on the early discovery of the condition, the degree of cardiac enlargement, the state of the myocardium and of the particular valves involved, the rhythm, the exercise tolerance, the education and preparation for a vocation with minimum additional damage to the heart, and the selection of a suitable vocation and harmless avocations.

Blackwater Fever following "Atebrin" Administration.

H. FOY AND A. KONDI (*Transactions of the Royal Society of Tropical Medicine and Hygiene*, June 25, 1937) record three cases of blackwater fever following the oral administration of "Atebrin" simplex. The patients had taken no quinine for months previous to their attack of hæmoglobinuria. They state that there is a general belief that "Atebrin" is free from the risks of precipitating hæmoglobinuria in potential blackwater fever patients. But these cases indicate that "Atebrin", like quinine and "Plasmoquine", is capable of precipitating hæmoglobinuria in certain patients. Whether these drugs are all equally potent in this respect remains to be determined. In two cases hæmoglobinuria occurred when the blood was devoid of parasites.

Fatal Asthma.

R. W. LAMSON AND E. M. BUTT (*The Journal of the American Medical Association*, May 29, 1937) discuss deaths from asthma. Recent insurance statistics indicate that the death rate from organic heart disease in asthma is three or four times the normal. These mortality rates are in sharp contrast with the observations of many clinicians, that the prognosis in asthma is excellent. In the literature, only about 50 case reports on patients who died of asthma have appeared in fifty years; 27 of the patients were males and 23 females. The average age at death of the males was 48 and of the females 43 years; but it was noted that prolonged severe asthma was not inconsistent with long life. The authors reviewed 137 additional patients who died in Los Angeles during the last eight years with a diagnosis of bronchial asthma. Of those on whom no necropsy was made the average age at death was 56·9 years for males and 49·7 years for females. Of those who came to necropsy the ages were 52 and 49. The authors point out that mistaken diagnosis is extremely common in so-called fatal asthma. Among their autopsy material coronary sclerosis, pleurisy, pneumonokoniosis and bronchopneumonia and heart disease were found in those certified to have died of bronchial asthma. Thirteen of these patients had heart disease apart from any asthma from which they may have suffered. In only 26% of all the patients was a diagnosis of allergic asthma justified. It is pointed out that insurance statistics, not based on *post mortem* findings, are unreliable in determining the death rate in such a disease as asthma.

"Tallqvist Anæmia."

WILLIAM DAMESHEK (*The New England Journal of Medicine*, November 18, 1937) states that the familiar lithographed Tallqvist scale is completely unreliable as a measure for hæmoglobin estimation. Many Tallqvist scales seem to give average readings of about 70%, and as a consequence "Tallqvist anæmia" is fairly prevalent and the cause of much unnecessary medication. The author has found it good policy to discard the scale completely. If a rough estimate is desired, comparison of the patient's palms with those of the physician is usually more satisfactory.

Prognosis in Coronary Thrombosis.

A. M. MASTER, S. DACK AND H. L. JAFFE (*Journal of the Mount Sinai Hospital*, November, 1937) have carefully studied three hundred cases of coronary thrombosis and have observed that the cardiac rate proved a reliable guide to prognosis. In patients whose heart rate did not go above 100 or below 40 per minute the mortality was 6%. When the rate was between 100 and 120 it was 29%. When the rate was between 120 and 150 it was 54%. However, only two out of nine patients who developed transient paroxysmal tachycardia died.

"Prostigmin."

UNDER the heading "Therapeutics of Prostigmin", L. S. Goodman and W. J. Bruckner (*The Journal of the American Medical Association*, March 20, 1937) record serious symptoms following the ingestion of 45 milligrammes of "Prostigmin" by mouth. The authors had taken doses of 7·5 milligrammes up to 30 milligrammes of "Prostigmin" orally at intervals of several days to avoid cumulative effects; finally 45 milligrammes were ingested three hours after luncheon. About three hours later excess nasal secretion was noted; this was followed by restlessness, defæcation, weakness and tremulousness. The hands and feet became cold, respiration was sighing and laboured, and there was a sensation of fluttering in the abdomen. Twitching of skeletal muscles was observed and became severe; there were several attacks of nausea, faintness, difficult breathing and a feeling of impending catastrophe. The pulse remained about 75 per minute, the tension being low. The pupils were contracted. Four hours after the drug had been taken, one one-fiftieth of a grain of atropine was injected intramuscularly; this was followed by gradual improvement, and within an hour recovery was complete. Consciousness was retained throughout. Intestinal peristalsis was observed during the attacks of faintness. Only one of the experimenters was affected by the "Prostigmin", the other having taken atropine earlier in order to prevent possible ill-effects. The authors point out the seriousness of the symptoms following oral administration of 45 milligrammes of "Prostigmin", and emphasize the danger of giving large doses in *myasthenia gravis*. They advise subcutaneous injection of 0·5 to 3·0 milligrammes of the drug thrice daily if necessary, in preference to oral administration, since it is not known whether the drug is cumulative when ingested.

Divinyl Ether.

I. S. RAVDIN *et alii* (*The Journal of the American Medical Association*, April 3, 1937) report observations of the use of divinyl ether as a general anæsthetic. Two thousand two hundred and sixty-six patients were anæsthetized by the open drop method and 409 by the closed method with nitrous oxide and oxygen or oxygen alone. For anæsthesia of longer than half an hour the method with oxygen is advised as less likely to cause liver necrosis. Caution is necessary to avoid over-concentration of the ether in the blood. Eleven to eighteen milligrammes per 100 cubic centimetres of blood are the average concentrations necessary for extraabdominal and abdominal operations respectively. Anæsthesia is rapidly induced and rapidly recovered from; muscular relaxation occurs quickly. The anæsthetic is mainly useful in children, because of the absence of struggling, for ophthalmic work, in reducing fractures, opening abscesses, and short periods of anæsthesia in the surgical outdoor department. It has replaced gas and oxygen for these purposes at the Hospital of the University of Pennsylvania. Only experienced anæsthetists should administer divinyl ether, induction being rapid, laryngeal irritation rare and excitement very unusual when the drug is skilfully given. Excessive mucus was noted in 79 patients, and cyanosis in six. Relaxation was satisfactory. Recovery was more rapid than with nitrous oxide. Vomiting occurred in 173 patients. Three patients who died within a week of operation were examined at autopsy. No ill-effects of anæsthesia were noted. Post-anæsthetic complications otherwise did not occur in this series. Divinyl ether is less toxic than diethyl ether, and more toxic than chloroform.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at Saint Vincent's Hospital, Sydney, on September 23, 1937. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Atrophic Vulvitis and Leucoplakia Vulvæ.

DAME CONSTANCE D'ARCY, in conjunction with DR. C. DE MONCHAUX and DR. NELL FARRAR, showed patients suffering from atrophic vulvitis and *leucoplakia vulvæ*. She said that two years ago, in Melbourne, Dr. Brian Swift had described his work on the treatment of *pruritus vulvæ* with dilute hydrochloric acid and vitamin *A*. She had decided to investigate some cases of this type.

Arrangements were made for every patient with vulvitis attending the radiotherapy clinic at Saint Vincent's Hospital to have a fractional test meal carried out. The results of these tests showed that all the patients had a deficiency of free hydrochloric acid in the gastric juice and many suffered from complete achlorhydria. Accordingly, all patients presented the characteristic symptoms of pruritus, inflammation with excoriation and piling up of cells. These patients had been treated for the past two years with dilute hydrochloric acid and cod liver oil. In the more severe cases, in which leucoplakia was present, or in which keratinized masses were piled up around the clitoris, healing was aided by the use of X radiation. In some few advanced cases vulvectomy had been necessary, and in all the cases in which the condition proceeded to the carcinomatous stage the block operation of removal of vulva and glands had been carried out. The earlier and milder cases were satisfactorily treated by Swift's method.

The first patient was a woman, aged fifty years, who was suffering from leucoplakia of the vulva, possibly malignant. On May 25, 1931, X ray therapy was carried out. On January 11, 1932, complete vulvectomy was performed. After this operation the pathologists reported that no malignant cells were visible in the section of the vulva. The lymph glands had metastasized, but the malignant cells appeared to have been considerably influenced by radiotherapy and were no longer active. On May 11, 1936, a fractional test meal was performed, and the total gastric acidity was reported as being very low.

The second patient was a woman, aged forty-six years, who suffered from leucoplakia of the vulva and pruritus. On September 25, 1936, a test meal was given. A decrease of free hydrochloric acid for the first hour was discovered, with an increase above normal after two and a half hours. On September 30, 1936, X ray therapy was started. On September 13, 1937, the patient was given dilute hydrochloric acid with pepsin in a mixture to be taken three times a day. She was also given cod liver oil twice a day.

The third patient was a woman, aged sixty-seven years, who suffered from cancer of the vulva and leucoplakia. There was an ulcer the size of a large almond and extensive leucoplakia. On July 3, 1935, X ray therapy was undertaken, and by September 23, 1935, the ulcer was healed, though some glands were palpable in the right groin. On October 28, 1935, the patient complained of very little irritation. No ulcer was present and no glands were palpable. On March 21, 1935, complete vulvectomy was performed. The patient was subsequently given iron, liver, hydrochloric acid with pepsin, and cod liver oil.

The fourth patient was a woman, aged forty-seven years, who was suffering from *pruritus vulvæ*. She was treated by dilute hydrochloric acid with pepsin and cod liver oil. At the same time X ray therapy was applied to the vulva.

The last patient was a woman, aged fifty-five years, who was suffering from leucoplakia of the vulva and pruritus. On January 16, 1935, X ray therapy was commenced. On June 1, 1936, it was discovered by the result of a test meal that the free hydrechloric acid in the gastric juice was considerably decreased. The patient was then given dilute hydrochloric acid with pepsin, and cod liver oil.

Von Recklinghausen's Disease.

DR. J. E. SHERWOOD showed a female patient, aged forty-four years, who complained of lumps all over the body, which had been present for as long as she could remember. Some of the lumps had increased in size recently. There appeared to be no family history of similar trouble. She had had four children, none of whom were similarly affected. She had had five miscarriages and three pelvic operations. Her previous medical history had been otherwise uneventful. Examination revealed the pigmentation and tumour formation characteristic of von Recklinghausen's disease.

Dr. Sherwood explained that von Recklinghausen's disease was a disease of congenital origin, characterized by cutaneous pigmentation, multiple neurofibromata, and often by secondary changes in the viscera and bones. The cutaneous fibromata were formed from the connective tissue elements of the cutaneous nerves. Neurofibromata might become sarcomatous. The disease was sometimes hereditary. Some of the symptoms were always present at birth, for example, cutaneous pigmentation. The pigmentation consisted of brownish spots, *café au lait* in colour, varying in size from a pin's head to areas the size of the palm. Cutaneous fibromata were soft, pinkish swellings, which might be sessile or pedunculated and varied in size from a pin's head to that of an orange.

Gout.

Dr. Sherwood also showed a male, aged thirty-three years, who complained of pain and swelling of the left great toe of two weeks' duration. His trouble actually appeared to have commenced four years previously. During this period an incision was made over a "cellulitis" in the same situation. Examination revealed tenderness, redness, swelling, and glossiness over the region of the metatarsophalangeal joint of the left great toe. Tophi were to be seen on both ears, especially the right.

Dr. Sherwood said that his excuse for showing the patient was that gout seemed to be becoming less frequent at the out-door department.

Juvenile Rheumatism.

Under the heading of juvenile rheumatism, Dr. Sherwood showed two girls, both fourteen years of age. One suffered from right-sided hemichorea with endocarditis, the other from a condition analogous to multiple infective arthritis of the rheumatoid type in an adult.

The latter patient complained of pains in both shoulders and in the knuckles of both hands of one month's duration. There had been no history of "growing pains" or sore throat. Tonsillectomy had been performed. On examination the interesting feature was the presence of the spindle-shaped proximal inter-phalangeal joints of both hands.

The former patient had been afflicted with irregular and purposeless movements of the right arm and leg for six years. The movements ceased during sleep. She had had one period of remission for twelve months after treatment. She complained of aching pains in the knees, wrists and elbows. There had been no history of sore throat. She had had tonsillectomy performed. Examination revealed a mitral systolic murmur conducted into the axilla. The right-sided hemichorea was very noticeable.

Congenital Heart Disease.

Dr. Sherwood also showed a male patient, aged nine months. He had been noticed to have prominent tips of the fingers of both hands and toes of both feet since he was three months of age. Examination revealed cyanosis of the lips and tips of the fingers of both hands and tips of the toes of both feet as well as clubbing of the fingers and toes. A blowing systolic murmur was audible over the tricuspid and mitral areas of the precordium.

Disseminated Sclerosis.

Dr. Sherwood's next patient was a man who first consulted him in 1934 when he was twenty-eight years of age. He complained of a painful and "paralysed" right leg.

giddiness, loss of sight in the right eye, and pains in the abdomen. All these symptoms were of seven years' duration. He had previously been told that he was suffering from stomach trouble and an "incurable complaint". On examination the chief features of interest were right-sided optic atrophy, nystagmus, exaggeration of both knee jerks, bilateral ankle clonus, double Babinski reaction. Both blood and cerebro-spinal fluid failed to react to the Wassermann test.

Subacute Combined Degeneration of the Spinal Cord.

Dr. Sherwood's last patient was a man, aged fifty-four years, who complained of epigastric pain of six years' duration. The pain occurred at any time, but more especially two to three hours after meals. He complained also of numbness of the fingers of both hands of six weeks' duration. He had had mumps some years previously. His father died from stomach trouble and had suffered from anæmia. His pupils—contracted and equal—reacted to light and accommodation. There was slight loss of power of both upper limbs with very little muscular wasting. His vibration sense appeared intact in both upper limbs. The finger-nose test gave unsatisfactory results. Both knee jerks were absent. The right ankle jerk was active, the left was absent. The plantar reflexes were "dubious". Slight Rombergism was present. The patient's vibration sense in both lower limbs was considerably disturbed. X ray examination of stomach and duodenum revealed no abnormality. The blood failed to react to the Wassermann test.

On July 16, 1937, the erythrocytes numbered 2,900,000 per cubic millimetre. The hæmoglobin value was 72% and the colour index 1·24. Pronounced anisocytosis, moderate poikilocytosis and slight polychromasia were present. The white cells numbered 6,000 per cubic millimetre, neutrophile cells being 70%, lymphocytes 23%, monocytes 5%, eosinophile cells 1%, and basophile cells 1%. On August 16, 1937, the erythrocytes numbered 3,800,000 per cubic millimetre, the hæmoglobin value was 86%, and the colour index 1·3. Moderate anisocytosis and slight poikilocytosis was present. The leucocytes numbered 8,000 per cubic millimetre. On September 13, 1937, the erythrocytes numbered 3,860,000 per cubic millimetre, the hæmoglobin value was 84% and the colour index was 1·10. Slight anisocytosis and very slight poikilocytosis were present. The leucocytes numbered 5,400 per cubic millimetre, and of these 68% were neutrophile cells, 26% were lymphocytes, 2% were monocytes, and 4% were eosinophile cells. The patient's serum did not react to the Wassermann test.

Skiagrams.

DR. D. G. MAITLAND showed a comprehensive series of X ray lantern slides of general interest; included amongst them were (*a*) lesions of the gastro-intestinal tract, (*b*) renal lithiasis and bilateral renal tuberculosis, (*c*) gas gangrene following compound fracture of the forearm, (*d*) Albers Schönberg's disease, (*e*) a series illustrating pregnancy and the diagnosis of intrauterine congenital deformities of the fœtus.

A MEETING of the Victorian Branch of the British Medical Association was held at Ballarat on November 27, 1937, the convenor of the Science Subcommittee, DR. A. E. COATES, in the chair. The afternoon meeting was held at the Ballarat Base Hospital and took the form of a series of clinical demonstrations by members of the honorary medical staff of the hospital.

Diseases of the Lung.

DR. G. T. JAMES showed three patients with pathological conditions of the chest. The first was a female, aged sixty-five years, who had first come under notice a year before the meeting, and it was considered probable that she was suffering from bronchogenic carcinoma. In June, 1936, when the cough had become noticeable, the condition was at first regarded as bronchitis. The cough had not been constantly present but had recurred intermittently. On three occasions the patient had had a hæmoptysis of one to two ounces of blood and intermittently at long intervals the sputum had been slightly blood-stained. Dyspnœa had never been present and she had been free of pain. She had reached a maximum weight of ten stone four pounds early in 1937, but during the past six weeks she had lost weight, and at the time of the meeting she weighed nine stone ten pounds. In 1929 cholecystectomy had been performed on this patient. Dr. James said that the sputum had been examined repeatedly but no tubercle organisms had been found. Examination of the blood serum had been negative as far as complement fixation tests for hydatid and venereal diseases were concerned, and the presence of slight secondary anæmia had been established by examination of the blood cells and hæmoglobin. A series of radiograms of the chest was demonstrated by Dr. James, and it was chiefly on the appearances in these films that the diagnosis of bronchogenic carcinoma had been advanced. Careful search for malignant disease elsewhere had been made, but no evidence was found.

Dr. James's second patient, a male aged forty-one years, had been referred to Dr. James at the chest clinic by Dr. Spring, of Ballarat. The patient had been working in a mine in the district at a 300-feet level, and it was stated that several other men had been affected similarly. Early in November, during work at the mine, the patient had had a severe coughing spasm, with a feeling like compression of the chest and difficulty in obtaining a satisfying inspiration. The condition had persisted for a week and he had continually coughed up huge quantities of inoffensive sputum without any unusual appearance, such as the presence of grape skins. Before the onset of these symptoms he had had a mild morning cough for some weeks, which had caused a certain amount of discomfort, but which had not been associated with the expectoration of much sputum. Dr. James stated that the patient had become quite well again and was able to ride a bicycle over country roads backwards and forwards to work without pain or discomfort, though he had a slight cough with very little sputum. The examination of his sputum for tubercle bacilli had revealed nothing on five occasions. A slight secondary anæmia was present; 12,700 leucocytes per cubic millimetre of blood had been estimated on the only occasion on which a white cell count was made, and Casoni intradermal and hydatid complement fixation tests had not been carried out. Dr. James demonstrated antero-posterior and lateral skiagrams of the chest.

The third patient shown by Dr. James was a female, aged thirty-two years, who had been confined at the end of her sixth pregnancy in August, 1936, the puerperium having been uneventful. At the end of the second week, however, after the extraction of a tooth, she had had an acute pulmonary infection with signs localized at the right base. The temperature chart had eventually settled to normal, but tachycardia had persisted with signs of enlargement of the thyreoid gland and toxicity. The patient had been kept under observation until December 17, 1936, but had not reappeared for examination until April, 1937. At that time she was coughing up foul sputum, and dulness was present over the base of the right lung. Two or three weeks later she had a fluctuant swelling posteriorly over the lower portion of the right side of the chest, and on May 25 Dr. Longden had drained the empyema with immediate improvement in the patient's condition. She had remained in hospital until September. The temperature became normal, the pulse rate varied between 85 and 90 beats per minute; the sinus was discharging very slightly and she was feeling very well and had regained her ordinary weight by the time she was discharged from hospital. At the time of the meeting she had lost a little weight and had some cough with expectoration, and she had had some intermittent mild febrile attacks with loss of appetite, malaise, slight shivers and a little discharge from the wound in the chest. Dr. James demonstrated the skiagrams which had been prepared and which showed that a large pulmonary abscess had been present.

DR. C. J. O. BROWN said that Dr. James had shown three very interesting lung cases. The diagnosis was difficult on the evidence submitted, and in each instance Dr. Brown

considered that bronchoscopic examination and radiographic examination after the introduction of lipiodol through the bronchoscope were procedures essential for accurate diagnosis. In the case of the first patient shown by Dr. James the abnormal shadow seen in the antero-posterior film might be due to one of many conditions, but in the lateral film the shadow could be seen to be due definitely to the lower lobe of the lung taking up a posterior position. Most of the shadow was caused by the presence of atelectasis of the lower lobe, which was most probably due to carcinoma of the bronchus blocking off that lobe. The accuracy of this opinion could be definitely determined by bronchoscopy and the introduction of lipiodol. After the respiratory passages had been sucked out the lipiodol might be able to pass the obstruction and the state of the breathing passages in the lower lobe would then be delineated in the film. Dr. Brown doubted if surgical treatment was possible. It was quite within the bounds of practical surgery to remove a lobe or for that matter the whole lung; but in most cases bronchogenic carcinoma was fairly centrally placed, and owing to the probability of recurrence the outlook was not bright. Dr. Brown illustrated the surprising length of such histories by referring to one patient in whom he had been able to satisfy himself that the condition had been causing symptoms for seventeen years before the patient died. He also said that bronchogenic carcinoma was often latent and unfortunately was quite common. It was liable to be found at autopsy to be the source of secondary carcinomatous deposits elsewhere. As an instance of this relationship he mentioned a quasi-primary cerebral carcinoma which had been found to be associated with a comparatively small primary bronchogenic carcinoma. He was satisfied that in these cases a large part of the shadow seen in the film was due to obstruction.

With reference to the radiograms of the second of Dr. James's three patients, Dr. Brown said that there was a localized thickening in the hilus, but no definite bronchial stenosis or atelectasis. From the appearances in the skiagrams he thought it was quite likely that a mediastinal tumour was present affecting glands in the hilus and causing some peribronchial density. If it was malignant, it might be carcinomatous or lymphosarcomatous. Dr. Brown expressed the opinion that the condition was not suitable for surgery, but that transitory improvement would probably be achieved by means of deep X ray therapy; the response to therapy would be of interest, though it would not be helpful to the patient in the end. Bronchoscopy and lipiodol radiography would assist greatly in more accurate localization.

Dr. Brown said that the third patient shown by Dr. James had an abscess following the insufflation of infected material, and that abscesses after tooth extraction were occurring more frequently. The films shown by Dr. James contained evidence of a considerable amount of negative pressure and collapse. The film after the introduction of lipiodol demonstrated that the lipiodol seemed to stop at the sinus; injection of lipiodol into the sinus followed by the preparation of a new film would help to define the relations of the abscess cavity to the respiratory passages. He mentioned that Dr. Athol Blaubaum had a number of films in which there were no signs of filling before bronchoscopic aspiration, but in which filling became evident and the outline of the bronchiectasis was demonstrated after bronchoscopy. Dr. Brown referred to a patient who in 1927 had contracted idiopathic empyema. The collection was drained, but recurred six months later, and the sinus did not close. The patient had been left with a sinus, and when he was seen in 1934 it was ascertained that every few months bleeding occurred from the chest sinus and the sputum contained blood. The patient became toxic and had a dry tongue in addition to anorexia, constipation and some fever. On removal of the overlying chest wall Dr. Brown had found thirty or forty bronchial fistulæ; the lower lobe bronchus terminated abruptly. The patient had been left with a great hole in the chest, but his general health had improved. He was still able to go about actively and he even went swimming. Dr. Brown stated that the bronchus was completely obstructed by carcinoma, which had certainly been there since 1927. In the case of Dr. James's patient, Dr. Brown suggested that the roof of the abscess should be removed to let it fill up from the bottom. The skin could be turned in at the edges of the wound, and it was always possible later to free the skin and turn it over the healing granulations.

DR. SYDNEY PERN referred to Dr. James's last patient as an example of the usual catastrophe after removal of teeth when the coughing reflex was lost. Dr. Pern was interested particularly in the sudden appearance of thyreo-toxic goitre, which illustrated the way in which acute infection produced toxic goitre.

(To be continued.)

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Alderdice, Alexander Arthur, M.B., B.S., 1936 (Univ. Sydney), 3, Neridah Street, Chatswood.

Medical Societies.

THE MEDICO-LEGAL SOCIETY OF SOUTH AUSTRALIA.

Programme for 1938.

ADDRESSES will be given at meetings of the Medico-Legal Society of South Australia, to be held at the Refectory, University of Adelaide, as follows:

On Friday, March 18.—"The Capacity to Make a Valid Will": H. Mayo, Esq., K.C., Dr. F. H. Beare, Dr. H. McI. Birch.

On Friday, June 17.—"The Liability for Negligence in Surgical Operations": G. C. Ligertwood, Esq., K.C., Dr. P. S. Messent, S. H. Skipper, Esq.

On Friday, September 16.—"Errors in Diagnosis": Dr. J. G. Sleeman, A. J. Hannan, Esq., K.C.

On Friday, November 18.—"Professional Confidence": Dr. C. T. C. de Crespigny, G. S. Reed, Esq., K.C.

H. A. McCOY,
J. L. TRAVERS,
Joint Secretaries.

Post-Graduate Work.

GENERAL REVISION COURSE IN SYDNEY, 1938.

THE General Revision Course will be held in Sydney under the auspices of the New South Wales Post-Graduate Committee in Medicine from April 4 to April 14, 1938. Application to attend the course should be made as soon as possible to the Secretary, New South Wales Post-Graduate Committee in Medicine, The University of Sydney, enclosing cheque and giving the following information: home address, Sydney address, whether railway concession tickets are desired.

The fee for the course will be £3 3s.; for mornings only or afternoons only, or for one week only, £2 2s.; for hospital residents and those not engaged in actual practice the fees will be half. Cheques should be made payable to the "Post-Graduate Fund in Medicine" and exchange added to country or interstate cheques.

Programme.

Monday, April 4.

Robert H. Todd Assembly Hall, 135, Macquarie Street.

9.15 to 10.30 a.m.—Registration.

10.30 to 10.45 a.m.—Official opening and address by the Minister for Health, the Honourable H. P. FitzSimons, M.L.A.

Morning tea.

11 a.m. to 12 noon.—"Some Points in Medico-Legal Autopsies": Dr. A. A. Palmer.
12 noon to 12.45 p.m.—"Practical Hints in Gynæcology": Dr. Ralph Worrall.
1 p.m.—The members of the course will be the guests of Sydney Hospital at luncheon.

Sydney Hospital (The Maitland Lecture Theatre).

2 to 3 p.m.—"Cutaneous Neoplasms", lecture-demonstration: Sir Norman Paul.
3 to 4 p.m.—"Pneumonia": Dr. A. Holmes à Court.
4 to 5 p.m.—"Varieties and Treatment of Acute Nephritis": Dr. Harold Ritchie.

Tuesday, April 5.

The Prince Henry Hospital.

9.30 to 10.30 a.m.—"The Clinical Significance of Blood Pressure Readings": Sir Charles Blackburn.
10.30 to 11.30 a.m.—"The Present Position of Surgery of the Thyreoid": Dr. H. R. G. Poate, Lecturer in Post-Graduate Surgery and Director of the Surgical Unit.
11.30 a.m. to 12.30 p.m.—"Arteriosclerosis": Dr. S. A. Smith, Lecturer in Post-Graduate Medicine and Director of the Medical Unit.
2 to 3 p.m.—"Gastroscopy": Dr. John Horan.
3.30 p.m.—Official opening of the Post-Graduate School of the Prince Henry Hospital.

Wednesday, April 6.

(Up to 9.30 a.m. on April 6 and 10.30 a.m. on April 7 demonstrations of cancer of the uterus will be given at various hospitals as part of the proceedings of the ninth Australian Cancer Conference.)

The Royal North Shore Hospital of Sydney.

9.30 to 10.30 a.m.—"Diabetes in the Young": Dr. W. Wilson Ingram.
11 a.m. to 12.45 p.m.—Practical demonstration of common pathological tests: Dr. Marjorie Little.
1 p.m.—The members of the course will be the guests of the Royal North Shore Hospital at luncheon.
2 to 3 p.m.—"Common Rectal Diseases and their Management": Dr. V. M. Coppleson.
3 to 4 p.m.—"Physical Signs in Diseases of the Chest"; demonstration of cases: Dr. Cotter Harvey.
4 to 5 p.m.—"Physiotherapy": Dr. A. L. Ducker.

The Robert H. Todd Assembly Hall.

5 p.m.—Cancer Conference. Cinematograph films of medical interest will be shown.

The Blaxland Galleries.

8 p.m.—Cancer Conference. Public address by Professor E. C. Dodds. (Tickets may be obtained from the supervisor.)

Thursday, April 7.

Saint Vincent's Hospital.

9.15 to 10 a.m.—"The Management of Head Injuries": Dr. I. D. Miller.
10 to 11 a.m.—"Gall-Bladder Infections": Sir John McKelvey.
11.30 a.m. to 12.15 p.m.—"The Treatment of Affections of the Mucosa of the Nose and Nasal Sinuses": Dr. J. J. Woodburn.
2.15 to 3 p.m.—"Inflammatory Lesions of the Eye", lecture-demonstration: Dr. Guy Antill Pockley.
3 to 4 p.m.—"Infections of the Hand": Dr. V. J. Kinsella.
4 p.m.—Cinematograph film of infections of the hand.

Friday, April 8.

The Royal Alexandra Hospital for Children.

9 to 10 a.m.—Removal of tonsils and adenoids (three theatres): Dr. H. Huff Johnston, Dr. N. Meacle and Dr. J. Steigrad.
10.15 to 11.15 a.m.—"Congenital Abnormalities of the Alimentary Tract": Dr. P. L. Hipsley.
11.30 a.m. to 12 noon.—"Selected Skin Cases": Dr. George Norrie.
12 noon to 12.30 p.m.—Selected surgical cases: Dr. T. Y. Nelson.
2 to 3 p.m.—"Infantile Paralysis": Dr. Edgar Stephen, Dr. Wilfred Vickers.
3 to 4 p.m.—"Clinical Signs and Types of Infantile Paralysis in the Victorian Epidemic" (illustrated by cinematograph films): Dr. Mostyn Powell, Medical Officer to the Government and Municipal Committee controlling the campaign against Poliomyelitis, Melbourne.

Saturday, April 9.

Sydney Hospital.

9.15 to 10.30 a.m.—Demonstration of fractures and their treatment": Dr. George Bell, Dr. A. Aspinall, Dr. A. M. McIntosh.
10.45 to 11.45 a.m.—"The Management of Genito-Urinary Diseases by the General Practitioner": Dr. Reginald Bridge.

Monday, April 11.

Lewisham Hospital.

9.15 to 10.45 a.m.—"Peptic Ulcer": Dr. J. D. Herlihy, Dr. Richard Flynn, Dr. Alan Oxenham.
11.15 a.m. to 12 noon.—"Differential Diagnosis between Cardiac and Renal Failure": Dr. C. G. McDonald.
12 noon to 12.45 p.m.—"Pitfalls in Anæsthesia": Dr. H. J. Daly.
1 p.m.—The members of the course will be the guests of the Reverend Mother and Community at luncheon.
2.30 to 3.15 p.m.—"Anæmia and its Treatment": Dr. Leo Flynn.
3.15 to 4 p.m.—"Intestinal Emergencies in General Practice": Dr. J. Kennedy.

Tuesday, April 12.

The Women's Hospital, Crown Street.

9.15 to 9.45 a.m.—"Treatment of Ante Partum Hæmorrhage": Dr. R. B. C. Stevenson.
9.45 to 10.15 a.m.—"The Use of Willett's Scalp Traction Forceps": Dr. H. C. E. Donovan.
10.15 to 10.45 a.m.—"The Management of Incomplete Abortion with Infection": Dr. J. Chesterman.
11 to 11.30 a.m.—"When and How to Interfere in Toxæmias of Pregnancy": Dr. R. McD. Bowman.
11.30 a.m. to 12 noon.—"Ante-Natal Recognition of Obstetric Difficulties", ward demonstration: Dr. A. J. Gibson.
12 noon to 12.30 p.m.—Demonstration of external version: Dr. T. Dixon Hughes.
Afternoon.—The Australian Golf Club, Rosebery: Golf match for the Post-Graduate Golf Cup.

Wednesday, April 13.

The Royal Prince Alfred Hospital.

9.15 to 10.15 a.m.—"The Treatment of Influenza and its Complications": Dr. Allan Walker.
10.15 to 11 a.m.—"Sulphanilamide in the Treatment of Gonococcal Infections": Dr. N. M. Gibson.
11.30 a.m. to 12.30 p.m.—"Sprains and Ligamentous Injuries of the Knee and Ankle", lecture-demonstration: Dr. L. G. Teece.
(Luncheon may be obtained at the refectory at The University of Sydney.)
2.15 to 3.15 p.m.—Gynæcological demonstration: Dr. H. Schlink.
3.15 to 4.15 p.m.—"Some Recent Advances in Medicine": Dr. A. J. Collins.

Thursday, April 14.

The Royal Hospital for Women.

9.15 to 10 a.m.—"Obstetrical Odds and Ends": Professor J. C. Windeyer.
10 to 10.30 a.m.—"Diagnosis and Treatment of Infection with Trichomonas Vaginalis": Dr. F. Brown Craig.

10.30 to 11 a.m.—"The Care of Premature Infants": Dr. Margaret Harper.
11.15 to 11.45 a.m.—"Rupture of the Uterus", demonstration: Dr. H. A. Ridler.
11.45 a.m. to 12.30 p.m.—"The Endocrines in Gynæcology": Dame Constance D'Arcy.

LECTURE IN MELBOURNE.

THE Melbourne Permanent Post-Graduate Committee wishes to announce that a lecture, "Unhappy Results in the Treatment of Fractures", will be delivered in the Medical Society Hall, 426, Albert Street, East Melbourne, by Dr. Kellogg Speed, B.Sc., M.D., Professor of Clinical Surgery, Rush Medical College, Chicago, Illinois, at 8.15 p.m. on Friday, February 25, 1938.

It is hoped that a film illustrating first aid treatment of fractures of the lower extremities will also be shown. The fee for admission will be five shillings.

Obituary.

JAMES BERNARD GUNSON.

WE regret to announce the death of Dr. James Bernard Gunson, which occurred on January 25, 1938, at Adelaide, South Australia.

JAMES VERNON McCREERY.

WE are indebted to Dr. Paul Ward Farmer for the following account of the career of the late Dr. James Vernon McCreery.

Dr. James Vernon McCreery was born at Kilkenny, Ireland, on October 10, 1843, and qualified as a surgeon at the Royal College of Surgeons, Ireland, in 1864, at the age of twenty-one years.

His father, who owned a property in the south of Ireland, gave him a letter to Dr. Lawlor, of the Dublin Lunatic Asylum, and he put in two or three months there as medical officer while waiting for a boat to Australia.

He sailed from Ireland in the *Queen of the North*, arriving in Victoria in 1865, and was registered here on August 4, 1865, being number 500 on the medical register of Victoria, and his is the oldest name on it.

For three years he assisted Dr. Crooke and Dr. Hewlett in Nicholson Street, Fitzroy. He then went to the goldfields at Okatiki, New Zealand, for six or seven months, but came back to Australia, and Dr. Paley, head of the lunatic asylums in Victoria, who resided at Yarra Bend, appointed him to the Ararat Asylum, which had been recently opened, as medical officer. This was in May, 1868. He stayed at Ararat for four and a half years, then came to Melbourne as senior medical officer and was stationed at Yarra Bend.

In 1876 Dr. Gordon, superintendent of Ararat, died. Dr. McCreery was sent to Ararat to take his place. He was transferred to Kew Asylum in 1883, at the age of forty, and stayed there until he left the department. He was appointed Inspector-General of the Insane of Victoria when Dr. Dick died in 1894, and remained in this position until 1905, when Dr. W. Ernest Jones was appointed. He founded the Idiot Asylum at Kew and carried it on against orders, as he was told to make it a ward of Kew Asylum, but made it into a department of its own.

His first wife was his cousin, Olympia McCreery, of Tasmania. There were two children, a son and a daughter, who survive him. His second wife was Miss Ella Boyes. There was one son of this marriage, and he was killed in a motor accident.

Dr. McCreery and Dr. John William Yorke Fishbourne, both Irishmen, did valuable pioneering work in the Lunacy Department.

I also worked in conjunction with him for twenty-five years in connexion with the Church of England Diocesan Mission to the Streets and Lanes of Melbourne, admirably conducted by the late Sister Esther.

The writer has pleasant memories of Dr. and Mrs. McCreery, who were both well-mannered; and about thirty-eight years ago Dr. McCreery did him a great service, and he only found out three or four years ago of this kind act. Therefore I mourn the loss of a dear friend.

Correspondence.

THE PROTECTION OF AUSTRALIAN ABORIGINES.

SIR: It is surprising that the most important factor in deciding native policy should remain in the background. This factor is skilled medical care of all natives in each category, tribal, mission and free. The latter class are those natives who live and work in white communities.

In their tribal state the natives of the far north of Australia are helpless victims of at least four endemic diseases: malaria, yaws, hookworm and ulcerative granuloma. All these diseases are curable by specific drugs. Untreated, each disease becomes an intolerable scourge.

For some years past too much prominence has been given to the views of anthropologists and too little has been heard of the accumulated knowledge of men like the Chief Protector at Darwin. However, the signs are obvious that authority has made up its mind, and one feels confident that the result will be to the future benefit of natives and Europeans alike. Controlled contact with white civilization will be the only solution of the problem.

At the present time the Cape York Peninsula and the Torres Strait islands can show examples of all stages of evolution, from bush tribes to civilized people. Gradually the bush natives are learning the value of agriculture and the comfort of housing and regular food during the wet season. They appreciate proper medical treatment, and their faith in the supernatural powers of a medical man is often embarrassing.

The missions, with the assistance of the Queensland Government, have done good work. Perfection is not yet. Nevertheless, the people of Australia should be grateful for what has been done and what will be done in future.

To those who still believe that natives should be left alone, I suggest that they should come and see for themselves. The "free" bushmen, crippled by yaws, present a sorry spectacle beside the healthy mission natives.

Yours, etc.,

JOHN R. NIMMO.

Thursday Island,
January 15, 1938.

MATERNAL MORTALITY.

SIR: I do not think that the humility of mind of the general practitioner should be so great as to accept, without demur, Professor Marshall Allan's remark that "Some of the results of maternal mortality may be ascribed to the emotion of fear which seems to supersede that of reason when an emergency arises". It would be as just, and is as unjust, as a similar accusation by the general practitioner against the hospital consultant when patients of the latter die in hospital.

As tackling emergencies is the business in life of the general practitioner, and the supersession of reason by emotion would quite unfit him (her) for practice of any kind, such an accusation should be withdrawn; it cannot be proved, it is quite false, and its acceptance by the teachers of the profession may lead to medico-legal actions against unfortunate practitioners.

The practitioner does not give his patient eclampsia and other toxæmias, contracted pelves, nor the autogenous

infections. He takes every precaution against carrying infection, and is at least as successful as the surgeon in preventing accidental infection. He reduces and does not cause the death rate; and until teachers as well as practitioners realize this we shall have a faulty outlook on the practice of midwifery, which prevents the profession achieving as good results as might otherwise be obtained; but it is the teacher, not the practitioner, who is responsible for this faulty outlook.

Yours, etc.,

MARY C. DE GARIS.

Geelong,
Victoria,
January 20, 1938.

DIATHERMY IN INDUSTRIAL SURGERY.

SIR: Mr. John Kennedy (THE MEDICAL JOURNAL OF AUSTRALIA, January 15, 1938) asks: "Is there any proof that any form of diathermy gives rise to more than hyperæmia of skin, and possibly subcutaneous tissues, in actual practice?"

I asked myself the same question fifteen years ago and satisfied myself there was by placing a clinical thermometer in the centre of the femoral shaft, in the middle of the liver mass, and between the cerebral hemispheres of an anæsthetized animal and then passing an appropriate diathermy current with pads applied externally to the shaven skin. There was in each case a very definite rise of temperature within five minutes of switching on.

Yours, etc.,

JOHN HOETS.

143, Macquarie Street,
Sydney,
January 20, 1938.

DEATH RATE IN MALARIAL THERAPY.

SIR: In an article on pyrexial therapy in the journal of January 8, 1938, reference was made to a 5% to 10% mortality in malarial therapy. In view of a somewhat widespread misconception as to the dangers of this form of treatment, I referred to the Department of Mental Hospitals of New South Wales, and have to thank Dr. Clifford Henry for the following information:

> Of the first hundred cases of general paralysis treated from 1927 onwards, four died within a month of the termination of malaria, and twenty-five within two months. One or two cases died while infected with malaria, but the figures are not immediately available.
>
> Of a hundred cases treated up to the end of last year, none died during malarial infection or within a month of the termination of treatment, but four died during the following month.

It is open to question how many of the deaths in this last series may be attributed to malaria. With a reasonable selection, and the rejection of advanced, rapidly advancing or emaciated cases, and those with myocardial failure, the risk from malarial therapy is negligible. Nor is there any need to allow the paroxysms to continue until the patient becomes profoundly exhausted. No elaborate technique is necessary to determine when the patient has had enough. It is, I think, fair to say that a patient who is not strong enough to stand malarial therapy will rarely be a good subject for one of the other forms of pyrexial treatment.

I should like to take this opportunity of quoting from Nicol and Hutten (*British Journal of Venereal Diseases*, July, 1937), who speak from experience of 800 cases of general paralysis of the insane treated by malaria, but who rejected another 200 as too advanced for treatment.

"We feel very strongly that the present unsatisfactory recovery rate in G.P.I. cannot be appreciably improved until it is recognised in this country that examination of the C.S.F. is as essential as examination of the serum." They found that one course of malaria was sufficient to produce a negative cerebro-spinal fluid in about 80% of their cases. But they state also: "we consider that the relatively small number (about 20%) of clinical recoveries which we obtain is due to the fact that irreparable damage is done to the brain by the spirochætes before the malaria therapy is given. Is this matter for surprise when patients receive varying and apparently useless kinds of treatment over still more varying periods of time, until finally they become psychotic?"

Early recognition of involvement of the nervous system will avail the patient little unless failure of the cerebro-spinal fluid to respond promptly to antisyphilitics is followed by some form of pyrexial therapy.

Yours, etc.,

W. S. DAWSON.

Sydney,
January 21, 1938.

The British Association of Radiologists.

FELLOWSHIP EXAMINATION.

Of the nine candidates who sat for the examination for the Fellowship of the British Association of Radiologists, held at the beginning of December, 1937, the following have satisfied the Fellowship Board:

F. Ellis, M.B., Ch.B. (Sheffield), D.M.R., R.C.P. & S. (honours in radiotherapeutics).

R. McWhirter, M.B. (Glasgow), F.R.C.S. (Edinburgh), D.M.R.E. (Cambridge) (honours in radiodiagnosis and radiotherapeutics).

E. L. Rubin, M.D. (Liverpool), D.M.R.E. (Liverpool) (honours in radiodiagnosis).

M. C. Tod, M.B., F.R.C.S. (Edinburgh), D.R. (Edinburgh) (honours in radiotherapeutics).

J. Z. Walker, M.B., Ch.B. (Glasgow), D.M.R.E. (Cambridge) (honours in radiodiagnosis).

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1925 to 1935*, of Queensland, as duly qualified medical practitioners:

Davies, Llewellyn Swiss, M.B., B.S., 1937 (Univ. Melbourne), Brisbane.

Day, Robert Sydney, M.B., B.S., 1934 (Univ. Adelaide), Bundaberg.

Dorney, Paul Laurence, M.B., B.S., 1937 (Univ. Melbourne), Brisbane.

Jackson, David Clements, M.B., B.S., 1937 (Univ. Melbourne), Brisbane.

Kurrle, Ernest Nicholls, M.B., B.S., 1937 (Univ. Melbourne), Brisbane.

McEwen, George, M.B., B.S., 1937 (Univ. Melbourne), Brisbane.

Parker, Alexander Hayward, M.B., B.S., 1936 (Univ. Sydney), Brisbane.

Picone, Dominic George, M.B., B.S., 1937 (Univ. Melbourne), Brisbane.

Pilkington, Reginald Ralph, M.B., B.S., 1937 (Univ. Melbourne), Chillagoe.

Rose-Innes, Arthur, M.R.C.S. (England), L.R.C.P. (London), 1917, Brisbane.

Wadeson, Geoffrey Laurence, M.B., B.S., 1937 (Univ. Melbourne), Brisbane.

Windsor-McLean, Leonard Allan, M.B., B.S., 1932 (Univ. Adelaide), D.T.M., 1935 (Univ. Sydney), Mount Isa.

Corrigendum.

IT was reported on page 134 of the issue of January 15 that Dr. D. D. Cade took part in a discussion on the insulin treatment of schizophrenia at a meeting of the Section of Neurology and Psychiatry of the Victorian Branch of the British Medical Association. Dr. Cade informs us that the remarks attributed to him were made by Dr. J. F. J. Cade, of Beechworth.

A WARNING.

MEMBERS are warned against giving help to a young man who states that he is a former medical student and a brother of a Melbourne doctor. He is not deserving of help, and it will save trouble if members heed this advice.

Books Received.

STORY OF A GREAT HOSPITAL: THE ROYAL INFIRMARY OF EDINBURGH, 1727-1929, by A. L. Turner, M.D., LL.D., Hon. F.R.C.P.; 1937. Edinburgh: Oliver and Boyd. Royal 8vo, pp. 421, with illustrations. Price: 10s. net.

TEXTBOOK OF GYNÆCOLOGY, by W. Shaw, M.A., M.D., F.R.C.S., F.C.O.G.; Second Edition; 1938. London: J. and A. Churchill Limited. Medium 8vo, pp. 614, with 4 plates in colour and 253 text-figures. Price: 18s. net.

THE RADIOLOGY OF PULMONARY TUBERCULOSIS, by J. E. Bannen, M.B., Ch.B., D.M.R.E.; 1937. London: Baillière, Tindall and Cox. Demy 8vo, pp. 163, with illustrations. Price: 12s. 6d. net.

THE COLLAPSE THERAPY OF PULMONARY TUBERCULOSIS, by J. Alexander, B.S., M.A., M.D., F.A.C.S.; 1937. London: Baillière, Tindall and Cox. Demy 4to, pp. 720, with illustrations. Price: 67s. 6d. net.

Diary for the Month.

FEB. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 10.—Federal Council Meeting, Melbourne.
FEB. 15.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 23.—Victorian Branch, B.M.A.: Council.
FEB. 24.—South Australian Branch, B.M.A.: Branch.
MAR. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAR. 2.—Western Australian Branch, B.M.A.: Council.
MAR. 2.—Victorian Branch, B.M.A.: Branch.
MAR. 3.—South Australian Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," pages xx to xxii.

BRISBANE AND SOUTH COAST HOSPITALS BOARD, BRISBANE, QUEENSLAND: Honorary Orthopædic Surgeon.

DEPARTMENT OF INSPECTOR-GENERAL OF HOSPITALS, SOUTH AUSTRALIA: Resident Medical Officer.

THE HOSPITALS COMMISSION OF NEW SOUTH WALES: Resident Medical Officers.

TOOWOOMBA HOSPITALS BOARD, TOOWOOMBA, QUEENSLAND: Resident Medical Officer, Relieving Medical Officer.

THE WESTERN SUBURBS HOSPITAL, CROYDON, NEW SOUTH WALES: Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135 Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178 North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

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